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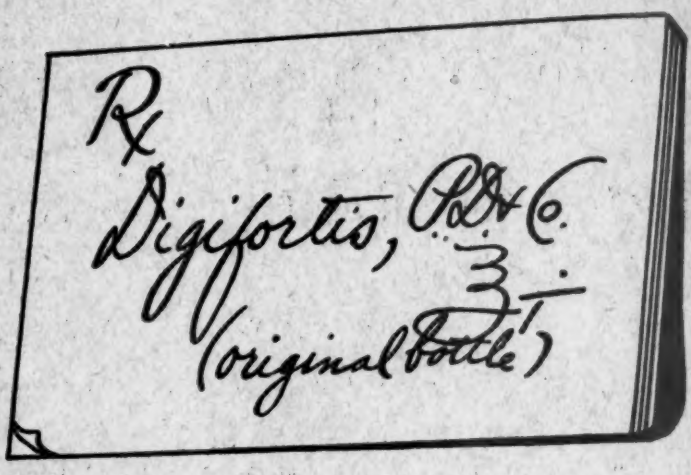

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PARKE, DAVIS & COMPANY

The American Heart Journal

VOL. I JUNE, 1926 No. 5

Original Communications

OBSERVATIONS ON FUNCTIONAL DISORDERS OF THE HEART*

PAUL D. WHITE, M.D.
BOSTON, MASS.

INTRODUCTION

FUNCTIONAL disorders of the heart are very common both with and without evidence of structural change in the heart itself. They are more frequently found without heart disease than with it. Generally the disturbances are so slight, due, for example, to effort syndrome or to premature contractions (extrasystoles), that medical advice is not sought, and their great frequency in the community is discovered only when groups of cases are questioned and examined for some routine purpose. Certainly it is true, however, that important disorders of cardiac function, like auricular fibrillation, heart-block, or congestive failure are found much more commonly in persons with evidence of structural change in the heart than in those without such evidence.

It is of considerable interest and importance to know what the likelihood of definite heart disease is in the case of a patient who consults a physician because of disturbed cardiac function. In order to determine this likelihood I have analyzed the records of 1,500 consecutive private patients whom I have seen during the past few years because of cardiac symptoms or signs. Of course, as has been said above, there are many people with functional cardiac disturbances who never consult a physician, perhaps being totally unaware of occasional premature beats or extrasystoles. Also there are patients ill with serious diseases other than heart disease who develop symptoms or signs of functional cardiac disorders, especially the effort syndrome. This is true particularly of infectious disease, such as tuberculosis and acute respiratory infections, and

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also of hyperthyroidism. Such cases naturally fall outside the scope of the present study, which includes only those individuals whose disorders of cardiac function or whose abnormal heart signs have been sufficient for them to ask advice for these disorders themselves. Of the whole group of 1,500 cases, 16 per cent showed congestive failure, many had fever, and 13 per cent had angina pectoris. This indicates that the entire group was composed of mixed kinds and grades of heart disease. It comprised all ages, and was about equally distributed between the sexes.

Thirty-six per cent (36.2 per cent) of the total series (543 of the 1,500 cases) had functional disorders of the heart without any demonstrable clinical evidence of cardiac structural change. This does not include a group of 48 cases of uncomplicated angina pectoris. If these be added to the functional list we shall have 591 cases or 39.4 per cent. The functional disorders shown by this group, excepting angina pectoris, were effort syndrome or the nervous heart, premature beats or extrasystoles of either ventricular or auricular origin, paroxysmal tachycardia, auricular flutter, auricular fibrillation, functional systolic murmurs and phobias of heart disease without other symptoms or signs. It is these disorders that form the basis of the present report. Angina pectoris will also be considered, since it is a common disturbance of function and often without clinical evidence of a cardiac lesion. Certain other functional disorders, namely, heart-block, pulsus alternans, and congestive failure, can be dismissed at once, because rarely, if ever, are they found except with structural changes in the heart, and then almost always they accompany very serious heart disease. Heart-block rarely may be produced in a normal heart by poisoning with digitalis; there were two such cases, both with effort syndrome, in the present series of 1,500 patients. Pulsus alternans and congestive failure may very rarely supervene in paroxysmal tachycardia of long duration and very high rate without organic heart disease. There were no such cases in this series.

Finally, in closing these introductory observations, it must be realized that cardiac disease may be present in some of the cases labelled "purely functional," but it is so slight in extent or so difficult to discover that it is overlooked and must be considered for practical purposes in a different category from that of organic cardiac disease that is readily found clinically.

EFFORT SYNDROME

The most common functional disturbance in the present series of cases is effort syndrome, sometimes called the "irritable heart," the "soldier's heart," or "neurocirculatory asthenia." The term "effort syndrome" is not very satisfactory, for excitement often acts like effort, or even more than effort, in producing the symptoms, which are usu-

ally palpitation, heart-ache, dizziness and even syncope. There are certain facts that always favor the diagnosis of effort syndrome, as opposed to organic heart disease alone, and these are a history of dizziness or fainting, the desire to take deep sighing respirations, an anxious state, overactive knee-jerks, and extreme sensitiveness to coffee.

The term "cardiac neurosis" I have reserved for a group of cases with only a fear or belief that the heart is diseased with no symptoms or signs at all. This term, however, as well as "the nervous heart," has often been used synonymously with effort syndrome.

The cause of effort syndrome is unknown, but the cardiac symptoms appear to be the result of a combination in various percentages of increased cardiac activity and of the victim's consciousness of this activity. The large majority of people with effort syndrome are of nervous temperament, that is, they appear to have inherited sensitive nervous systems. They have developed their symptoms almost always as the result of some nervous strain or infection. Those who have not yet learned how to live properly to control their trouble are inclined to rush both their work and their play. They live what is considered in other countries the typical high speed American life until "effort syndrome" makes them pause. Gradually the truth dawns upon them that they must slow up and sit and dream more, and try "to do" less. In this way they outgrow the trouble to a certain extent, but they are always liable to have a recurrence if the strain becomes heavy again, especially the strain of hurry and worry. They probably never really outgrow the likelihood of effort syndrome; they simply live and learn and gradually develop a philosophical attitude toward it, especially if they receive early the proper medical advice and reassurance. *Thus effort syndrome is dependent upon two factors: (1) the inherited nervous sensibility, and (2) nervous or physical strain. If neither factor is appreciable, there will be no effort syndrome; if both are pronounced effort syndrome will be marked in degree, while if either one or the other is negligible the remaining factor must be extreme to produce the symptoms.* The prognosis for life is excellent, of course, and for usefulness and happiness it is dependent upon the degree of nervousness plus the control of strain in the individual case. *There is no evidence as yet that it leads to organic heart disease later in life,* but the condition itself may, with sufficient provocation, recur at any age and, as is readily understood, may even occur for the first time in old age if the stage be set at that time. In old people naturally one must be more suspicious that organic heart disease may be present to cause symptoms, but effort syndrome is by no means simply a disorder of youth. It is even possible that effort syndrome, by forcing the subject to lessen the strain of work and play in youth, may retard the development of serious heart disease and arteriosclerosis later on. In the course

of time we shall begin to be able to gather reliable statistics on this point, while we watch the effect of age on many men who were victims of effort syndrome during the war, and on the men and women whom we have seen with the condition clearly recognized since the war.

The treatment of the condition is obvious and generally consists in a full discussion with the patient of his problem, his inherited nervousness, his manner of life and the need of reducing the strain on his nervous system if the symptoms are severe. Mild symptoms may probably be ignored if they do not increase. *Reassurance wins at least half the battle, but also a full recognition of the discomfort and disability must be granted by the physician.* Only thus can he gain the confidence of the patient, who, without such frank discussion of medical ignorance as well as knowledge, tends to wander from one doctor to another, or finally to Christian Science healer or chiropractor, where he may eventually find relief. Drugs are generally quite unnecessary, even the bromides, except in rare cases, while some, like digitalis, are quite harmful and increase the symptoms. Rest in bed is unnecessary and also often harmful, resulting now and then in confirmed invalidism. After the war there were victims of effort syndrome who should have been at work, but instead were living as helpless invalids because a diagnosis of "myocarditis" had been made in their cases. This has been bad not only for them, but has proved a great burden for the government and all of its taxpayers.

There were 220 cases of uncomplicated effort syndrome in the series of 1,500 patients, 15 per cent, or almost one-half of all those with functional disturbances alone; 100 were in males and 120 in females. There were only 13 under the age of twenty years; 60 were between twenty and thirty years of age; 54 between thirty and forty years, and 56 between forty and fifty years, many of these last being women at the time of the menopause. There were but 37 over fifty years of age. I was impressed by the small number (13) under twenty years of age, and the fairly even distribution of cases between twenty and fifty-five years.

In addition to these 220 patients there were 65 more "functional" cases with effort syndrome, 39 complicated by premature beats or extrasystoles, 20 by paroxysmal tachycardia, 5 by functional systolic murmurs, and 1 by marked sinus arhythmia, making a total of persons with effort syndrome without organic heart disease of 285 (or 19 per cent of the 1,500 cases). Of the cases with premature beats 19 were in males and 20 in females; of those with paroxysmal tachycardia 6 were in males and 14 in females; of those with prominent functional systolic murmurs one occurred in a man and 4 in women, and the one case of effort syndrome with marked sinus arhythmia was found in a woman,

making a total of all the groups of 126 males and 159 females. The age incidence of the cases of effort syndrome with premature beats was as follows: one between ten and twenty years, 8 between twenty and thirty, 15 between thirty and forty, 11 between forty and fifty, and 4 between fifty and sixty; that of the cases of effort syndrome with paroxysmal tachycardia was: 2 between ten and twenty years, 3 between twenty and thirty, 9 between thirty and forty, and 6 between forty and fifty; that of the cases of effort syndrome with functional systolic murmur was: one between ten and twenty, 2 between twenty and thirty, one between thirty and forty, and one between forty and fifty; while the patient with effort syndrome and marked sinus arhythmia was twenty-four years old. Thus the age incidence in the groups of combined functional disorders with effort syndrome is essentially the same as in the uncomplicated group of 220.

It is important to remember that effort syndrome may occur with organic heart disease and produce all or most of the symptoms in such cases, which are then frequently wrongly suspected of having congestive failure. In the present series of 1,500 cases there were 31 patients with well marked effort syndrome complicating rheumatic heart disease, 15 complicating hypertensive heart disease, 13 of them women at the time of the menopause, 10 complicating arteriosclerotic heart disease, 3 with angina pectoris, one with congenital heart disease, and none among the uncommon luetic aortitis patients.

Thus there were 60 cases of pronounced effort syndrome in combination with organic heart disease (4 per cent) as contrasted with 285 of effort syndrome cases without evidence of organic heart disease (19 per cent). This means a total incidence of effort syndrome of 23 per cent. *From the figures here compiled the chances that the patient coming to the physician with symptoms of effort syndrome will show no evidence of organic heart disease are approximately five to one.*

PREMATURE BEATS (EXTRASYSTOLES)

The next most common functional disorder in the series was found to be that due to premature beats or extrasystoles. As has been already emphasized, many persons with premature beats never consult the physician because they are unconscious of the arhythmia or because they at least are not concerned about it. But of those individuals with this disorder uncomplicated, who did seek medical advice, there were in this series 76, 46 male and 30 female; only 2 were under twenty years of age, 8 between twenty and thirty years, 15 between thirty and forty, 20 between forty and fifty, 16 between fifty and sixty, and 15 over sixty years of age. There were also the 39 cases of premature beats complicating effort syndrome already mentioned: 19 in males and 20 in females, with a considerably lower age incidence. One patient was between ten and twenty years of age; 8 were between twenty and

thirty; 15 between thirty and forty, 11 between forty and fifty, and 4 between fifty and sixty years. Twenty-eight cases with paroxysmal tachycardia without evidence of organic heart disease also showed premature beats and 4 cases with paroxysmal auricular fibrillation without evidence of organic heart disease had premature beats. There were thus altogether 147 "functional" cases with premature beats, 84 in males and 63 in females.

Etiological factors in the production of the extrasystoles in these cases were often impossible to discover, but occasionally there seemed to be a clear relationship between fatigue, tobacco, indigestion, or mild or obscure infection and the occurrence of the arhythmia. If the cause was evident, its correction and reassurance of the patient were satisfactory therapeutic measures; reassurance was always helpful. Drugs were usually ineffective when tried—digitalis, quinidine, and bromides.

In 103 cases, of the 1,500, premature beats were found with organic heart disease, 58 in males and 45 in females, with a considerably higher age incidence than in the case of premature beats without organic disease. There were 2 cases under ten years of age, none between ten and twenty, 4 between twenty and thirty, 7 between thirty and forty, 11 between forty and fifty, 23 between fifty and sixty, 34 between sixty and seventy, 20 between seventy and eighty, and 2 over eighty years of age. Seventeen of these cases also had paroxysmal tachycardia and 13 had paroxysmal auricular fibrillation. *The average age of the cases with organic heart disease showing premature beats was fifty-eight years, as contrasted with that of forty-three years in the cases without organic heart disease, an average of fifteen years older for the "organic" group.*

It is of some interest to ascertain the relative occurrence of auricular and of ventricular premature beats. An electrocardiographic study of 71 of the cases, 36 without and 35 with evidence of organic heart disease, showed 13 cases with auricular premature beats, 8 of them in the "organic" group; 53 cases with ventricular premature beats, 30 of them in the "functional" group, and 5 cases with both auricular and ventricular premature beats, 4 of them in the "organic" group. Thus ventricular premature beats occurred in 58 cases in all, and auricular premature beats in 18.* *From these figures it may be said that five-sixths of the cases with premature beats without evidence of heart disease show extrasystoles of ventricular origin only, while the remainder have auricular extrasystoles; a rare case shows both. Of the cases with premature beats and evidence of heart disease two-thirds show extrasystoles of ventricular origin; the rest have auricular extrasys-*

*Of 761 individuals with premature beats electrocardiographed at the Massachusetts General Hospital, the large majority of whom had organic heart disease, 245 (or 32 per cent) showed auricular premature beats only, 504 (or 66 per cent) showed ventricular premature beats only, and 16 (or 2 per cent) showed both. There were also 92 patients with auricular fibrillation who showed ectopic ventricular contractions.

toles; in over 10 per cent both types are found. Premature beats tend to occur in greater numbers in an "organic" than in a "functional" case, but there are frequent instances where, without any evidence of other heart trouble, extrasystoles are frequent, even occurring every second or third beat over considerable periods of time (hours or days).

Thus *there were altogether in the series of 1,500 cases 250 with premature beats (17 per cent), of which 147, or about three-fifths, did not show organic heart disease, and 103, or two-fifths, did show organic heart disease.*

PAROXYSMAL TACHYCARDIA

Among the "functional" cardiac cases in the series, paroxysmal tachycardia comes third in frequency. There were 89 patients with paroxysmal tachycardia without evidence of organic heart disease. Twenty-eight of them showed premature beats also and twenty others had well marked effort syndrome. Of the eighty-nine cases, 36 were in males and 53 in females. The age incidence was as follows: none under ten years of age, 7 between ten and twenty years, 12 between twenty and thirty, 28 between thirty and forty, 23 between forty and fifty, 18 between fifty and sixty, and one over sixty years of age. Probably very few cases of paroxysmal tachycardia are missed, as compared with the many missed cases of premature beats, for it is indeed a rare individual who is not conscious of, and at least to some degree concerned about, paroxysms of tachycardia which usually last from a minute or two to several hours and cause the heart rate to increase to from 120 to 200 or more per minute. The etiological factors, when discovered, were much the same as in the case of premature beats: fatigue, indigestion, recent infection, and so on. The treatment consisted in attempts to get rid of the exciting factor, reassurance, and the successful use of quinidine sulphate in occasional cases, either by rations to prevent or reduce the number of attacks, or in larger doses to stop the attacks. Vagal or ocular pressure or some other special measure like the Valsalva experiment may sometimes abolish an individual attack.

In comparison with the 89 "functional" cases with paroxysmal tachycardia there were 43 cases with paroxysmal tachycardia showing evidence of organic heart disease, 14 in males and 29 in females, with an age incidence almost ten years higher than in the "functional" group, averaging forty-eight years as compared with thirty-nine years. *Seventeen of the organic heart disease group with paroxysmal tachycardia were over sixty years of age, while only one of the functional cases with this abnormal rhythm was more than sixty.*

Thus of the total number of 132 patients with paroxysmal tachycardia, 43 gave evidence of organic heart disease and 89 did not. *It may be said from these figures that the chances that a patient with paroxysmal tachycardia has no evidence of heart disease are about two to one*

AURICULAR FLUTTER

Auricular flutter is a rare disorder of the heart and was found in only 8 cases of the 1,500 (½ of 1 per cent), 5 with and 3 without evidence of organic heart disease. Of those with evident heart disease 2 patients were male and 3 female, and their ages were, one between twenty and thirty years, one between forty and fifty, two between fifty and sixty, and one over seventy. Of the three "functional" cases, one patient was male and 2 female, one between twenty and thirty, and 2 between thirty and forty years. The cases without evidence of organic heart disease closely resembled the "functional" cases with paroxysmal tachycardia.

AURICULAR FIBRILLATION

Auricular fibrillation is a common and serious functional disorder of the heart almost always found with evidence of organic disease. The relative rarity of its occurrence without evident cardiac pathology makes one hesitate somewhat to consider that it may be possible for it to occur solely as a functional disorder. In this respect it closely resembles angina pectoris, but there is no question but that, like angina pectoris, it may be found without definite evidence of organic heart disease. Perhaps in such cases there are cardiac lesions (for example, coronary sclerosis) which are not demonstrable clinically.

In the present series of 1,500 cases there were 198 patients with auricular fibrillation (13 per cent), 170 with evidence of organic heart disease (86 per cent), and 28 without (14 per cent), a ratio of seven to one. There are two main types of auricular fibrillation, the paroxysmal and the permanent. In the paroxysmal fibrillation group there were 65 cases out of the total of 198 (33 per cent), and in the permanent fibrillation group there were 133 cases (67 per cent).

A. Paroxysmal auricular fibrillation.—Of the 65 patients with paroxysmal auricular fibrillation 15 were without and 50 with evidence of organic heart disease, a ratio of 3 to 10. Of the "functional" group, 12 were males and 3 females, the age incidence being as follows: 2 between twenty and thirty years of age, 3 between forty and fifty, 5 between fifty and sixty, and 5 between sixty and seventy. Of the 50 cases with evident organic heart disease 25 were in males and 25 in females, with a higher age incidence than in the "functional" group: 2 between thirty and forty years of age, 8 between forty and fifty, 10 between fifty and sixty, 21 between sixty and seventy, 6 between seventy and eighty, and 3 over eighty years. The average age of the patients with paroxysmal auricular fibrillation with no evident cardiac pathology was fifty-two years, while that in the patients with evidence of organic heart disease was sixty-one years. Much the same factors seemed to be responsible for the production of the paroxysms of auricu-

lar fibrillation as for attacks of regular paroxysmal tachycardia, fatigue, indigestion, infection and sudden, very vigorous exertion. Because the paroxysms of auricular fibrillation tend to last longer than regular paroxysmal tachycardia, often twelve hours or more, they usually are more disabling and demand more attention. The patient may be reassured, but at the same time he is often very much helped by the use of quinidine sulphate, both in small doses as daily rations (for example, 3 grains by mouth 3 or 4 times a day), and in larger doses at the beginning of a paroxysm to cut it short (for example, 6 grains at once and 6 grains repeated twice, if necessary, at two-hour intervals). It is often in paroxysmal auricular fibrillation either with or without evidence of organic heart disease that spectacular relief may be afforded by the proper administration of quinidine sulphate. Paroxysmal auricular fibrillation in itself is often very disturbing but of little importance otherwise unless the paroxysms are very long or precipitate heart failure. If such complications ensue and normal rhythm has not quickly been restored by quinidine sulphate, digitalis must be resorted to, but the routine treatment of all cases of paroxysmal auricular fibrillation by digitalis either during or between the paroxysms is unjustifiable.

B. Permanent auricular fibrillation.—Of 133 patients with permanent auricular fibrillation in the present series, there were but 13* without definite evidence of organic heart disease. *Thus the chances are, from these figures, at least ten to one that a patient with permanent auricular fibrillation* will show clinically definite cardiac structural changes. Of the 13 "functional" cases 8 were in males and 5 in females, the age incidence being: one between twenty and thirty years of age, 3 between forty and fifty, 5 between fifty and sixty, and 4 between sixty and seventy. It seems likely that coronary sclerosis is present as a causative factor in some of these cases, as in some of those of the paroxysmal type, because of the rather high age incidence, averaging fifty-three years. Of the 120 cases with organic heart disease and constant auricular fibrillation, 59 were in males and 61 in females, with the age incidence as follows: 2 between ten and twenty years of age, 5 between twenty and thirty, 11 between thirty and forty, 22 between forty and fifty, 32 between fifty and sixty, 33 between sixty and seventy, 13 between seventy and eighty, and 2 over eighty years. There were many relatively young patients in this group, because of the frequent association of mitral stenosis and auricular fibrillation. The average age, however, of the whole group with constant auricular fibrillation and organic heart disease was fifty-five years.

The prognosis in the cases of constant auricular fibrillation without evidence of organic heart disease is generally excellent. Their ventric-

*Slight enlargement was found in 2 or 3 of these cases later by x-ray.

ular rate is usually high, however, and must either be controlled by digitalization and maintenance of digitalization, or else by restoration of normal rhythm, for which this group is especially fitted. Certainly every one of these patients should receive a satisfactory trial with quinidine sulphate before he is doomed to a routine life with the constant use of digitalis. I have had several instances, in this group of 13 "functional" cases with permanent auricular fibrillation, of restoration to normal rhythm and maintenance of normal rhythm for several years to present date with rare or no relapses, and only little need or use of quinidine after the original course.

ANGINA PECTORIS

Angina pectoris is certainly an expression of disturbed cardiovascular function, no matter what its special mechanism may be. Its paroxysmal nature and its relief by vasodilating drugs strongly suggest vasoconstriction as the cause; the close correspondence in position and type of pain between angina pectoris and acute coronary thrombosis or embolism (where there is proof of obstruction in the coronary circulation) makes it likely that the vasoconstriction, in large measure at least, involves the coronary arteries. Many patients with angina pectoris show no evidence of organic heart disease clinically, and such patients, if they fail to give their histories, can easily pass examination for life insurance. It seems likely that the patients with angina pectoris who do not show evidence of organic heart disease should, if they take good care of themselves, have a better prognosis than the others. This point in the prognosis of angina pectoris is now under investigation and will be reported shortly in another communication.

There were 188 cases of angina pectoris in the series of 1,500 (12½ per cent). Forty-eight of these (25.5 per cent) showed no evidence of organic heart disease. Of this "functional" group of 48, 35 cases were in males and 13 in females; the age incidence was as follows: one between twenty and thirty years of age, 2 between thirty and forty, 9 between forty and fifty, 26 between fifty and sixty, 9 between sixty and seventy, and one between seventy and eighty, giving an average age of fifty-four years. Of the 140 cases of angina pectoris with evidence of organic heart disease, 106 were in males and 34 in females. The age incidence was as follows: 12 patients between forty and fifty years, 38 between fifty and sixty, 63 between sixty and seventy, 26 between seventy and eighty, and one over eighty, giving an *average age of sixty-three years, nearly ten years greater than the group without organic heart disease.*

FUNCTIONAL SYSTOLIC MURMURS

Twenty-eight cases of the 1,500 were examined because of the finding at apex or base of a slight to moderate systolic murmur apparently not associated with valvular heart disease or other structural cardiac abnor-

mality. The mechanism of the production of these murmurs is obscure. Some of the louder ones in the region of the pulmonic artery are accentuated in the recumbent position by full expiration and thus appear to be associated with increase in intrapulmonary blood pressure. It is probable that lack of proper tone in the mitral valve ring sometimes explains the functional mitral systolic murmur, which of course becomes very marked with muscle weakness due to severe anemia. Very loud systolic murmurs often mean organic heart disease, particularly if they are accompanied by thrills and a history of rheumatic infection or probable congenital heart disease. Of the 28 cases referred for functional systolic murmurs in this series, 21 were in males and 7 in females; most of them were young, 4 being under ten years of age, 6 between ten and twenty, 7 between twenty and thirty, 6 between thirty and forty, 4 between forty and fifty, and only one over fifty years. There were also 5 cases already referred to with marked effort syndrome and functional systolic murmurs, one in a male and 4 in females, 3 under thirty years of age and all under fifty.

CARDIAC NEUROSIS

Some individuals develop a dread of heart disease, or belief that it is present, either because of the knowledge that friends or relatives have had heart trouble, or because they have themselves fainted or felt dizzy, or because some one has told them that they probably have heart disease. They have no cardiac signs or symptoms. Thirty-two cases of this nature were seen in this series, 19 in males and 13 in females, all but 2 between twenty and sixty years of age, and fairly evenly distributed. Reassurance after careful examination is all that is needed.

SINUS ARHYTHMIA

Marked sinus arhythmia as a cause for medical consultation is rare and such consultation generally results from the finding of a slow irregular pulse during convalescence from some infectious disease in youth. It is of no importance. It usually occurs in nervous individuals. There were seven cases altogether in the present series (0.5 per cent), one of them with well marked effort syndrome. Four patients were male and three female. The ages were nine, twelve, fourteen, fourteen, sixteen, twenty-four, and twenty-nine years. It should be pointed out here that sinus arhythmia is not the sign of a healthy heart. It is more often found in pronounced degree in young people, and young people are less apt to have heart disease than old people. Sinus arhythmia is common in young people with valvular disease of rheumatic origin, but it is absent, or at least very rare, in the presence of acute or subacute endocarditis, or of congestive heart failure; therefore its presence is helpful as evidence against those conditions. It is commonest in association with sino-auricular bradycardia and tends to be

exaggerated by the action of digitalis. It is easily increased in degree by forced respiration, which may be resorted to in establishing the diagnosis in doubtful cases. Reassurance is all the treatment needed.

SINO-AURICULAR TACHYCARDIA

Six patients in the series were seen simply because of a rapid pulse (0.4 per cent). No organic heart disease was found. Nervousness, or mild infection or slight hyperthyroidism were the apparent causes. There were 3 males and 3 females, aged seven and one-half, ten, thirty-six, thirty-eight, forty and forty-one years.

SINO-AURICULAR BRADYCARDIA

Three cases in the series were seen simply because of a slow pulse (just under a rate of 50 per minute). Two patients were female and one was male. They were fifteen, twenty-six and thirty-five years of age. Electrocardiograms showed no heart-block. Such bradycardia is not rare in many people early in the morning before rising, but it is uncommon in the middle of the day. It sometimes occurs during convalescence from infectious diseases. It is of no moment.

SUMMARY

An analysis of 1,500 consecutive patients with cardiac symptoms and signs, seen in private practice, has been made in order to determine the absolute and relative incidence of functional disorders of the heart. The figures are recorded.

Of those functional disturbances more often found without than with clinical evidence of organic heart disease in this series, effort syndrome is most common and occurs five times more often in the "functional" cases; premature beats sufficient to attract attention come next; and finally paroxysmal tachycardia, which is found twice as often without as with clinical evidence of organic cardiac disease. Functional systolic murmurs alone, cardiac phobias, marked sinus arhythmia, sino-auricular tachycardia and sino-auricular bradycardia are less often seen but together make an appreciable percentage of the whole series.

Of those functional disturbances more often found with than without clinical evidence of heart disease, auricular fibrillation (paroxysmal as well as constant) and angina pectoris are the most common and important. Both of these conditions do occur occasionally, however, without any obvious cardiac disease. Auricular flutter is rare. Heart-block, congestive failure, and pulsus alternans rarely if ever occur without evidence of serious heart disease.

CARDIAC NEUROSIS ASSOCIATED WITH RHEUMATIC VALVULAR HEART DISEASE*

L. E. Viko, M.D.
Salt Lake City, Utah

CARDIAC neurosis is not infrequently mistakenly diagnosed and treated as organic heart disease. Various writers[1, 2] have discussed the danger of misinterpretation of the symptoms and signs—especially murmurs—occurring in these variously named neuroses. But as White[3] and Herrick[4] have pointed out, cardiac neurosis and organic heart disease may coexist in the same patient, adding to the difficulty of diagnosis and treatment. This association of conditions is in contradiction to the usual definition for cardiac neurosis, where the absence of physical signs of structural diseases is given as essential for the diagnosis of neurosis. Yet when we consider these cardiac neuroses from the psychiatric standpoint as "functional mental disorders—attended primarily by cardiac symptoms,"[3] not only is there no contradiction in terms but it seems reasonable to suppose that in individuals with a certain mental make-up, organic heart disease might frequently precipitate and condition a neurosis. This combination of diseases is not rare yet seems to have had little discussion in medical literature. The present report deals with 23 such cases where cardiac neurosis was associated with rheumatic valvular heart disease.

SYMPTOMATOLOGY AND DIAGNOSIS

The symptoms presented by the 23 cases of this group were principally those of cardiac neurosis and the objective findings those of rheumatic heart disease. This was shown strikingly by comparing these cases with a group of 100 cases of valvular heart disease without neurosis and a group of 50 cases of cardiac neurosis without organic lesion, a comparison which also served in retrospect as a check upon the accuracy of the diagnosis.

In the comparison of the symptomatology of the three groups, there was presented a marked contrast between the valvular cases with neurosis and the valvular cases without neurosis.

Though 30 per cent of the cases with organic disease alone did not complain of palpitation, dyspnea or precordial pain, 100 per cent of the valvular-neurosis group complained of one or more of these symptoms. Only 9 per cent of the valvular cases complained of dizziness, fainting, paresthesia or choking (here grouped arbitrarily as

*Read before the American Heart Association, Dallas, Texas, April 20, 1926.

TABLE I

SYMPTOMS IN THREE GROUPS OF CASES

SYMPTOMS	VALVULAR 100 CASES	NEUROSIS 50 CASES	VALVULAR AND NEUROSIS 23 CASES
"Cardiac Symptoms" (one or more)			
Dyspnea, palpitation, precordial pains	70%	98%	100%
"Neurocirculatory Symptoms" (one or more)			
Dizziness, fainting, paresthesia, choking	9%	24%	87%
Sweating, nervousness, fatigue	14%	38%	55%
Phobias	0%	4%	13%
Irregularity of the heart	9%	17%	26%
Symptoms referred to other organs with recognized organic cause	39%	14%	17%
Without recognized organic cause	2%	54%	65%

"*neurocirculatory*" symptoms); 87 per cent of the cases where the valvular disease was complicated by neurosis presented one or more of these symptoms. Only 14 per cent of the former group noted nervousness, sweating or fatigue, while 55 per cent of the latter group were so affected. In both groups there were complaints referred to other organs than the heart, but, while in the uncomplicated valvular cases an organic cause for symptoms could usually be found, where neurosis was a complicating factor such symptoms were usually without organic basis. Considering the "*neurocirculatory* symptoms" separately (Table II), it was noted that while dizziness was recorded in only 7 per cent and fainting in only 3 per cent of the uncomplicated valvular cases, these symptoms were found in 74 per cent and 34 per cent, respectively, of the mixed group.

TABLE II

SYMPTOMS IN THE THREE GROUPS OF CASES

SYMPTOMS	VALVULAR 100 CASES	NEUROSIS 50 CASES	VALVULAR AND NEUROSIS 23 CASES
Dyspnea	62%	76%	91%
Palpitation	44%	86%	91%
Precordial pains	20%	52%	52%
Dizziness	7%	40%	74%
Fainting	3%	26%	34%
Paresthesia	4%	18%	38%

Similar differences in the frequency of disturbances of sensation were noted (Table II). The contrast was so marked that the occurrence of any of these symptoms in a case of valvular heart disease should lead to a suspicion of the presence of a complicating neurosis.

From the standpoint of symptomatology, then, the cases of valvular heart with neurosis were characterized, in contrast with the uncomplicated valvular cases, by a multiplicity of symptoms and by the great frequency of those symptoms here termed *neurocirculatory*,

for the want of a better term. In this they resembled the group of pure cardiac neurosis but showed such symptoms in even greater degree.

The objective findings in the 23 cases of the mixed group agreed with those in the uncomplicated valvular group. (Table III.)

TABLE III

OBJECTIVE FINDINGS IN THE THREE GROUPS OF CASES

	VALVULAR	NEUROSIS	VALVULAR AND NEUROSIS
Enlargement of the heart		0%	
Slight or none	52%		57%
Moderate or marked	48%		43%
Valve lesions	100%	0%	100%
Arhythmia			
Premature beats	7%	24%	4%
Paroxysmal tachycardia	0%	8%	0%
Auricular fibrillation	15%	0%	4%
Decompensation	26%	0%	4%

In the mixed group all presented definite valvular lesions varying from simple mitral regurgitation to mitral stenosis with or without aortic lesions. Ten of them showed moderate or marked enlargement of the heart; one had auricular fibrillation; this one only had been decompensated at any time, while of the uncomplicated valvular group, 26 per cent were definitely decompensated. Considering the cases of the neurosis-valvular group individually, there was a constant and striking lack of proportion between the symptoms and the objective evidence of heart impairment—a most important point in the diagnosis of these cases. If not obviously evident, this lack of proportion was brought out by prolonged observation of the patient and by comparison of the symptoms with x-ray and electrocardiographic studies of the heart and exercise tolerance tests. Even the case with auricular fibrillation and past history of decompensation exemplified this point.

Dejerine's criteria[5] for the diagnosis of neurosis in general emphasizes the illogical combination of symptoms in these cases. In most of the cases of this group, this was evident and served to point to the complicating neurosis. One girl could go through a dozen dances without discomfort and yet, often, far less exertion produced distressing symptoms. Another patient could, on one day, swim a good distance and on the next, without obvious change in the heart, be unable to sit up in bed without marked dyspnea. Usually emotion and not exertion induced symptoms in these cases; improvement or regression, too, was commonly coincident with the varying emotional states rather than with the amount of physical activity. In most of the cases, though the organic lesion may have been of long standing,

the more acute symptoms could be traced to an emotional rather than to a physical event—to a period of worry, a death in the family, war experience, maternal anxiety or, frequently, the patient's first knowledge of the organic lesion, or treatment for it.

The final characterization of these patients was reaction to treatment. The average patient with uncomplicated valvular heart disease resents or resists restriction of his activity. These patients with a complicating neurosis welcomed restriction and resisted efforts to increase their activity.

THE NEUROSIS AND ITS RELATION TO THE ORGANIC DISEASE

Classification of cardiac neurosis, as of neurosis in general, is neither easy nor entirely satisfactory. Using the classification of White and Cobb,[3] the cases of this series were divided as shown in Table IV.

TABLE IV

TYPE OF NEUROSIS—23 CASES

Anxiety neurosis	5
Fatigue neurosis (neurasthenia)	13
Introspection neurosis (hypochondria)	3
Substitution neurosis (hysteria)	1
Doubtful	1

A variety of underlying and precipitating causes have been assigned to the cardiac neurosis. One or more such factors could be elicited in most of the cases of this group. (Table V.)

TABLE V

ETIOLOGY OF THE NEUROSIS

Neurotic family history	6 cases
Neurotic personal history	5 "
Sex conflicts	2 "
Mental and emotional strain	4 "
Illness	2 "
Injury	1 "
War experience	1 "
Suggestion	
Maternal anxiety	3 "
From the doctor	8 "
Doubtful	6 "

Five had been definitely of the neurotic type since childhood. Various emotional factors had been present. Definite sex conflicts were discovered in only two cases—perhaps a psychiatrist would have shown this element to be more important. Acute infections did not seem to have played a large part. Chronic focal infections were present in seven of the cases and, though they were eradicated as part of the treatment, their relative importance could not be determined.

In general most of these etiological factors differed too little from such conditions in simple cardiac neurosis to merit extended discussion. It was rather the relation of the neurosis to the organic lesion that was of interest.

Probably in all cases the rheumatic heart disease antedated the actual cardiac neurosis. Five of the patients were so evidently of a neurotic type that quite probably some type of neurosis would have developed even in the absence of organic heart disease. In these and perhaps others the heart lesions may only have served to fix the major neurotic symptoms on the heart. Van Lier[6] has discussed this from the standpoint of the psychiatrist. In many of the cases, however, the connection between the organic disease and the neurosis seemed more direct; knowledge of the heart defect or treatment of it seemed the principal precipitating factor for the neurosis.

In three cases in children, maternal anxiety regarding a heart "leakage" in her child led to overrestriction and constant suggestion to the child. Two of these boys, with apparently normal make-up in the beginning, had at first strenuously combated efforts to restrict their sports but later developed an introspection type of neurosis. In one of these the physician had foreseen the outcome and endeavored unsuccessfully to combat the maternal anxiety. The third case was of the musical, dreamy sort and readily gave in to the maternal fears but was fortunately saved by a change of environment away from the mother.

In all three of these cases the neurosis was far more disabling than the organic lesion is ever likely to be. In these cases it would seem that the neurosis might have been prevented by less attention to the heart and more to the mind of the child.

In eight other cases an important factor was the diagnosis or treatment of the valve lesion by a doctor, whose unintentional suggestion seemed to serve as the starting point for the neurotic symptoms. In some cases, no doubt, the patient found the suggestion despite the doctor's proper judgment and treatment of the heart. In others a failure to recognize the neurotic element in the case, a mistaken judgment regarding the significance of some symptom, psychic rather than physical in origin, or an error in estimating the seriousness of the organic lesion led to the development of the acute neurotic symptoms, usually of the anxiety type. Particularly did this occur if the suggestion regarding the heart came to the patient during a period of emotional distress. One case illustrated this better than detailed discussion.

Case of Mrs. R. J., aged thirty-six years. Family history good; nervous and fearful as a child. Rheumatic mitral disease from childhood, without symptoms. At age of eighteen was told by doctors that she had heart trouble and must be careful; from this time had palpitation when nervous but not on active physical

exercise. In summer of 1923, while under prolonged nervous strain, developed dyspnea and attacks of dizziness, and numbness; doctor consulted advised restriction of physical activity. Symptoms increased. In December, father died suddenly of heart disease. Immediately after patient began having fainting spells. Doctor advised absolute rest in bed and gave digitalis. An acute anxiety neurosis followed with extreme fear of immediate death and great dyspnea, palpitation and weakness.

Cases of this type illustrate the importance of considering the mental make-up of any patient with heart disease, lest improper interpretation of symptoms in a neurotic person lead to a mental reaction to the lesion far worse than those due to the lesion itself. As this is not always easy, it seems better, if in doubt about the importance of a rheumatic lesion and if there be any neurotic tendency, to err on the side of too little treatment than on the side of too much.

The importance of recognizing the neurosis was evident from the fact that five of this group had been continuously confined to bed by the neurosis for from one to three months. All were in some degree incapacitated, and did not lead the normal life that the organic disease would have permitted.

Jaquet[7] and Swan[8] have described, in cases of organic heart disease, nervous and psychic distrubances which they considered due to circulatory disturbances of the cerebral cortex or other organs secondary to heart disease. In none of the present cases did it seem that such a mechanism explained the neurosis. All except one were well compensated, and in this one the neurosis was of a definite conflict type. The degree of neurotic symptoms was not proportional to the amount of cardiac impairment. If cerebral congestion had been responsible for the nervous symptoms, the treatment instituted should have increased them; this was not the result obtained.

RESULTS OF TREATMENT

Treatment in these cases was of necessity directed toward both the organic lesion and the neurosis; difficulty lay in the fact that treatment for one was in part antagonistic to that for the other. It was often a problem to determine what degree of activity might be safely forced on the patient—how complete a reassurance might be given him. Careful and frequent observation was necessary, not only for the safety of the patient but to relieve the apprehension in one's own mind that a mistake in judgment was being made. The results after periods varying from a few months to four years are shown in Table VI.

On the whole the results seem to have borne out the diagnosis and justified the treatment. In the most satisfactory cases the patient, convinced of the neurotic origin of the major symptoms, made a striking recovery. On the other hand in three cases, the neurosis

TABLE VI

RESULTS OF TREATMENT

THE ORGANIC DISEASE		THE NEUROSIS	
Improved	9 cases	Apparently cured	5 cases
Unchanged	7 "	Greatly improved	3 "
Worse	2 "	Improved	7 "
No reexamination or report	5 "	Worse or unchanged	3 "
		No reexamination or report	5 "

remained unchanged despite treatment. Two of the cases showed regression from the standpoint of the organic lesion. One of these was the case with auricular fibrillation referred to previously, in which both the organic lesion and the neurosis were so pronounced that little could be done for either. In the other case the neurosis was cured but the organic condition became worse, probably because the psychotherapy gave the patient too optimistic a viewpoint regarding the organic lesion. As a result, for a year there had been no medical advice sought and auricular fibrillation had ensued. This illustrates one of the difficulties in the treatment of such cases.

SUMMARY

1. Twenty-three cases are reported where cardiac neurosis was associated with valvular heart disease.

2. Suggestion from an anxious mother in three cases and from a doctor in eight cases were important factors in the development of the neurosis. Recognition of these nervous manifestations is of great importance.

3. The recognition and treatment of these cases is important and difficult.

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(*For discussion see page* 649.)

PAROXYSMAL VENTRICULAR TACHYCARDIA*

A Report of Five Cases

A. R. Gilchrist, M.B., M.R.C.P. (Ed.)
Edinburgh, Scotland

PAROXYSMAL ventricular tachycardia is regarded as one of the rarest of the cardiac arhythmias, but, with the increasing use of the electrocardiograph, cases are now being recognized more frequently, and the significance of the condition is better understood. It is not proposed to review fully the literature on this subject, as this has been done recently by Wolferth and McMillan.[1] In reporting their four cases, they refer to twenty-two others.

During the past two years, out of a total of eighty-five cases of organic heart disease of many types and in varying degrees of failure under treatment in the wards of this Department, paroxysmal tachycardia was recognized to be present on at least twenty occasions among eight patients. In four of these, electrocardiograms demonstrated that the paroxysms recorded were frankly of the ventricular type. This suggests that this form of tachycardia is less uncommon than the number of reported cases would indicate. Excluding their fifty-three cases of auricular flutter, Willius and Barnes,[2] in a series of forty-nine cases of paroxysmal tachycardia, found six of the ventricular type.

The five cases reported here are of interest in that they illustrate not only differences in the cardiac mechanism during the attacks, but also in their causation. This latter factor appears to be of importance from the point of view of the prognosis and treatment of the condition.

CLINICAL CASES

Case 1.—Mrs. M. A., aged fifty-two years, was a patient from 4/28/25 until her death on 7/14/25. She gave a history of five attacks of rheumatic fever, the first at the age of twenty, but admitted no cardiac symptoms until two months previous to hospital treatment, when an irritating cough began to bother her. This was followed rapidly by dyspnea on exertion, and later by orthopnea and a copious, white, frothy sputum. Three weeks previous to admission, edema of the ankles appeared, and in the course of a day or two spread up to the hips and back. Since then both hands had swollen at times, and the abdomen had become more and more distended. Examination revealed a "water-logged" patient, pale and cyanosed. There was generalized edema, which reached enormous proportions in the legs, and a tremendous amount of ascites, with enlargement of the liver. Heart was regular in rhythm, 150 per minute, auricular flutter was present, with marked venous engorgement of the superficial veins. The transverse diameter of the heart in the fourth space was 19 cm.

*From the Department of Therapeutics, University of Edinburgh, Scotland. Work done under the Medical Research Council.

with a ticktack gallop rhythm, and with mitral and tricuspid incompetence. There was no hydrothorax but marked congestion of both pulmonary bases. The urine contained a trace of albumin. Blood Wassermann reaction was negative. The vital capacity was 800 c.c., less than 28 per cent of the normal (West's[3] standards). The venous blood pressure was 28 cm. of blood (Young's[4] method). A massive dose of digitalis reduced the heart rate to 75 (4 to 1 flutter) without improvement in the patient's general condition. A few days later the rhythm changed to auricular fibrillation, and finally normal rhythm returned fourteen days after the single mas-

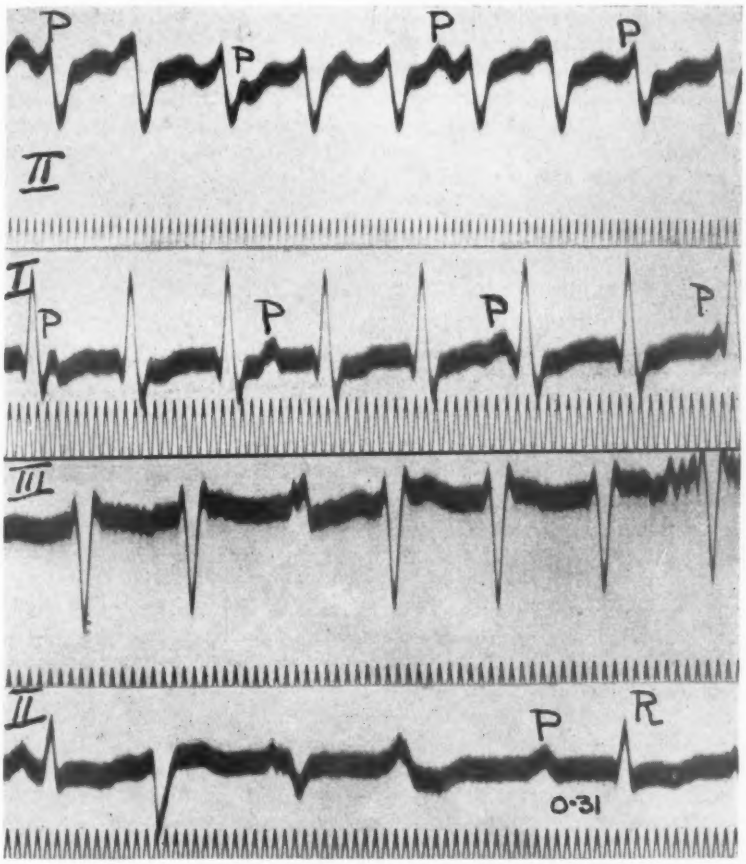

Fig. 1.—(Case 1.) Upper record,* (lead 2, 2 cm. 1 millivolt) taken on first day of paroxysm, shows the abnormal type of complex with dissociated auricular activity. The ventricular rate is 170 and the auricular 73 per minute.

Twenty-four hours later—two middle records (leads 1 and 3)—the paroxysm still continued. The P-waves are well marked in lead 1. Lead 3 shows a right-sided contraction replacing the third beat. The ventricular and auricular rates are now 170 and 76 respectively per minute.

The lowermost record (lead 2) taken on the day after the cessation of the paroxysm shows the return of normal rhythm with numerous extrasystoles succeeded by defective A-V conduction—the long P-R interval is 0.31 second.

*Time-marker. 28.57 vibrations equals 1.0 second. 1 cm. equals 1 millivolt in all records unless otherwise stated.

sive dose of digitalis had been given. Novasurol induced a slight diuresis, but improvement was not maintained. Later, small doses of Guy's pill twice daily were ineffective during a week's trial. This was followed by 0.4 gm. pulverized digitalis daily. Three days after commencing this preparation extrasystoles were noted, and

a single short run of pulsus bigeminus. Her condition seemed better and the digitalis was continued for three more days when paroxysmal ventricular tachycardia appeared, the attack lasting for nearly thirty-six hours with a ventricular rate of 170 (Fig. 1). Digitalis was stopped, and electrocardiograms, taken on the cessation of the paroxysm, showed normal rhythm with numerous extrasystoles, frequently in groups and followed by delayed A-V conduction. Her condition was now definitely worse, slight jaundice was present, and as all the usual diuretics were without avail, venesection and Southey's tubes to the legs were employed. Pulsus bigeminus returned, and daily the extrasystoles became less and less frequent as normal rhythm persisted. Ten days before her death she had a series of convulsions, each of which lasted from one to five minutes and was characterized by rapid clonic movements of the extremities, deviation of the head and eyes to the left, and in the intervals much mental confusion and transient attacks of aphasia. The heart rate during the fits was 140 or more, regular, but the type of rhythm present could not be ascertained. The legs became septic and she died peacefully on 7/14/25. A postmortem examination was not obtained.

Electrocardiograms from this case are shown in Fig. 1. The diagnosis of ventricular tachycardia depends upon the evidence of independent and rhythmic auricular activity during the paroxysm, and upon the close resemblance between the ventricular complexes with extrasystoles recorded before and after the paroxysm. That digitalis was the provoking cause seems evident from the fact that coupled rhythm preceded and succeeded the paroxysm, and that the premature beats became progressively less numerous after the drug had been discontinued.

CASE 2.—R. M., male, aged sixty-four years, was a patient from 12/4/24 until his death on 2/27/25. There was no history of rheumatic fever, but he had always been a heavy drinker, consuming whisky and beer daily for years. His occupation required much muscular activity, and for the last two years he had found it increasingly difficult. Giddiness, breathlessness on exertion, nocturnal frequency of micturition, and precordial pain had become increasingly severe during the past two years, and, for a few weeks previous to admission to hospital, edema of the ankles and ascites had been present. Examination revealed the presence of auricular fibrillation with advanced congestive failure. The apex beat was in the sixth space 16½ cm. to the left. There was marked arteriosclerosis, with albuminuria, casts and urinary output of 300 to 1000 c.c. per twenty-four hours. Blood urea was 36 mg. per cent, and phenolphthalein excretion was 11 per cent in two hours. Blood Wassermann reaction negative; vital capacity, 1,600 c.c., approximately 37 per cent of the normal. The patient was treated with various diuretics, with digitalis, and the usual dietetic measures, without improvement. In all, he received four courses of digitalis therapy. The first consisted of 0.4 gm. pulverized digitalis daily for five days. Two days later the heart rate had fallen to 62, with no improvement in his condition. The drug was then stopped. Sixteen days later he received a massive dose of pulverized digitalis (1.4 gm.), and this was followed two days later by a short paroxysm of tachycardia, lasting half an hour. A further massive dose (1.4 gm.) was given sixteen days after the previous one, and this was followed by two paroxysms of tachycardia, with a ventricular rate of approximately 150. Electrocardiogram of the second attack is shown in Fig. 2. It lasted for one hour and a half. Digitalis was discontinued for a fortnight, and as its repetition in small daily doses again invoked many brief paroxysms, its further use was therefore precluded. He died later, in advanced cardiac failure. A postmortem examination was not made.

Electrocardiograms from this case are shown in Fig. 2. In two separate attacks electrocardiograms were secured. In the others, cardiographic proof of their nature could not be obtained. The diagnosis of ventricular tachycardia is based upon the abnormal type of ventricular complex present (resembling extrasystoles) and upon the continuation of auricular fibrillation throughout the paroxysm. The paroxysms bore a definite relation to the administration of digitalis.

CASE 3.—D. M., male, aged sixty-one years, was a patient from 12/4/24 until his discharge from hospital on 4/17/25. As a young man he had suffered from relapsing fever, dysentery and "ague", influenza more recently, and three or four attacks of asthma. There was no history of rheumatic fever or alcoholic excess. He was brought to hospital unconscious, with the statement that he had had several con-

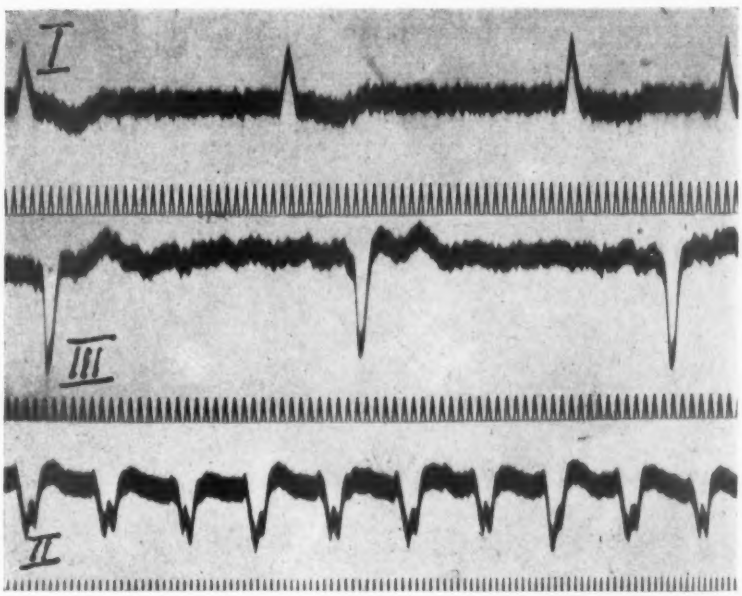

Fig. 2.—(Case 2.) The upper two records (leads 1 and 3) show auricular fibrillation present throughout the period during which the patient was under observation. Lower record (lead 2) was taken during a paroxysm of ventricular tachycardia. Irregular auricular waves can be seen between the complexes. The ventricular rate is 158 and the complexes vary in form from beat to beat.

vulsive seizures that morning. The same day he had three short seizures characterized by loss of consciousness, twitchings of the right side of the body, and followed by mental confusion. One similar attack had occurred eighteen months previously. On the day after admission he developed a left-sided hemiparesis, which was only transient, and disappeared the following day. He gave no history of dyspnea, palpitation, or edema. While under observation he had no further seizures, and the heart rate was regular, averaged 80, and no disorder of rhythm was noted except on one day, when the ventricular rate suddenly rose to 200. This paroxysm of tachycardia lasted for about one hour. The only symptoms which might be attributed to this attack were a feeling of uneasiness and slight palpitation. Apart from this, there were no abnormal physical signs detected on physical examination of the heart. There was, however, marked arteriosclerosis, with a faint trace of albumin. The

blood Wassermann reaction was negative. The vital capacity was 2,250 c.c., approximately 60 per cent of normal. He had not had any of the digitalis preparations. A postmortem examination was not made.

The electrocardiograms of this case are shown in Fig. 3. The paroxysm of tachycardia is presumably of the ventricular type, in that the complexes are of abnormal form. There is indefinite but strong presumptive evidence of auricular activity at half the ventricular rate, i.e., alternate blocking of retrograde impulses to the auricle. This is seen more clearly in lead I. The record (Fig. 3), taken the following day, shows normal rhythm with misshapen ventricular complexes and flattened T-waves, the decrease in potential of the deflections being suggestive of myocardial exhaustion and coronary sclerosis.

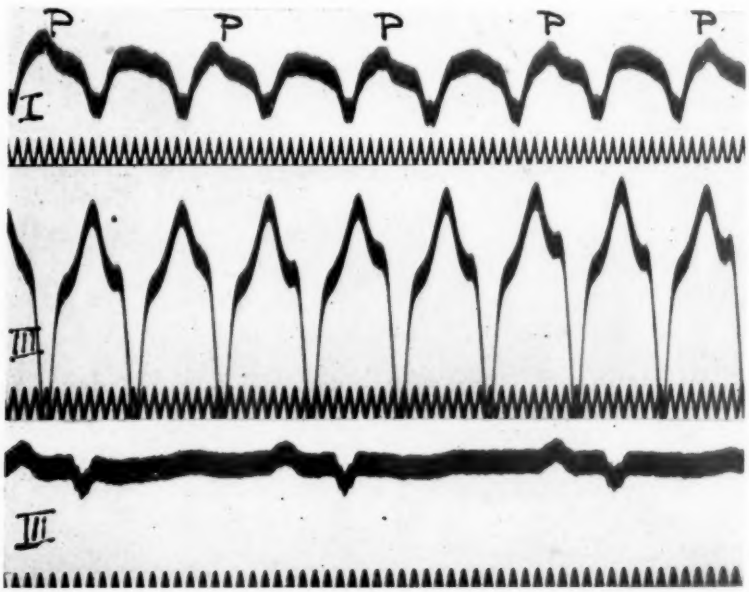

Fig. 3.—(Case 3.) The upper two records (leads 1 and 3) were taken during the paroxysm. P-waves are seen in lead 1 occurring at half the ventricular rate. This suggests retrograde conduction from the ventricle, alternate impulses being blocked. The ventricular rate is 210 and the auricular 105 per minute.

The lower record (lead 3) was taken after the cessation of the attack and shows normal rhythm with complexes of low potential. The heart rate is now 78.

CASE 4.—J. L., male, aged forty-one years, was a hospital patient from 7/9/23 to 7/25/23. There were no previous illnesses to note. His complaint was of short and abrupt attacks of palpitation brought on by exertion. He had noticed this for a year but was able to continue his work (mining) until a week before admission to hospital. He then found that while he was at his work he had a hot flushing sensation which came on suddenly, generally over the face and head, but sometimes going down the legs. He experienced dizziness on rising up suddenly from a stooping position. For the first time he became breathless, and had to rest at his work. The breathlessness was often brought about by only a trifling amount of exertion. A tight feeling was experienced across the chest, and he had been troubled by feeling the rapid beating of his heart. It was his habit for years to drink a pint of beer

daily and to smoke 5½ oz. of "black-twist" per week. He also chewed the same tobacco. Patient was a well-developed, muscular man, pale, but with no edema or cyanosis. The heart was slightly enlarged to the left. There were no murmurs heard. Blood pressure 120 mm. No arteriosclerosis. The blood Wassermann reaction was negative. The urine contained no abnormal constituents, and further physical examination was negative. When first seen, the pulse was irregular in rate, about 107, and ventricle about 150. Electrocardiograms were taken (Fig. 4), and four hours later, when the tachycardia had passed off, the heart rate was 70 with occasional extrasystoles. He had no further attacks while under observation and was discharged from hospital in good health. He reduced his tobacco consumption to 1½ oz. per week. On account of the anemia he was given ferri et quinin citrat. and instructed to report should attacks recur. He has not done so and attempts to trace this patient since his discharge from hospital have been unsuccessful.

The electrocardiograms from this case are shown in Figs. 4 and 5. The reasons for assuming the paroxysms to be of ventricular origin will be discussed later. Normal rhythm, present after the paroxysm had passed off, is shown in Fig. 5.

CASE 5.—W. M., aged nineteen years, was under observation from 10/20/25 until 3/14/26. There were no previous illnesses to note, and he had always been healthy until eighteen months previous to admission to hospital, when he began to suffer from transient attacks of giddiness and weakness, brought on by exertion. Each attack lasted less than five minutes, and seemed to be aborted by stooping forward and to the right side. One year ago he was experiencing three or four attacks a day, each characterized by giddiness, tinnitus, facial sweating, with palpitation of sudden onset and abrupt cessation. During the past six months, attacks had been more frequent and of longer duration, up to two or three hours, accompanied by dyspnea, and occasionally orthopnea at night. He sometimes felt as if he were "walking on air," vision became obscured, and he was forced to rest at his work until the palpitation ceased, when he felt better immediately. Latterly he has suffered from a sense of constriction across the chest during the attack, and after the attack tenderness, of several days' duration, was experienced in the region of the apex beat and anterior axillary fold on the left side. He was admitted to hospital with a heart rate of 150, dyspneic and in an exhausted condition. There had been no signs of congestive failure. On examination the heart was not enlarged, and no murmurs were heard, but the heart action was quite irregular. Tracings showed that this was due to an intimate mixing of short ventricular paroxysms, multiple and isolated extrasystoles and occasional beats of normal origin. The vital capacity at this time was 81.8 per cent. The blood Wassermann reaction was negative. The urine contained no abnormal constituents.

During the period the patient was under observation he had four attacks sufficiently severe to produce dizziness, weakness and general discomfort without evidence of congestive failure. His general condition improved materially, but trivial exercise continued to produce brief paroxysms and multiple extrasystoles. Digitalis was relatively ineffective and greater benefit followed the use of quinidine. His vital capacity rose from 81.8 per cent to 95.6 per cent during the time he was in hospital. Taking 7 grains of quinidine a day he has had no further attacks and is now able to undertake duties which formerly induced attacks.

The electrocardiograms from this case are shown in Figs. 6, 7, 8 and 9. The state of the heart on admission is shown in Fig. 6. A short paroxysm (with respiratory tracing) during which bilateral ocular

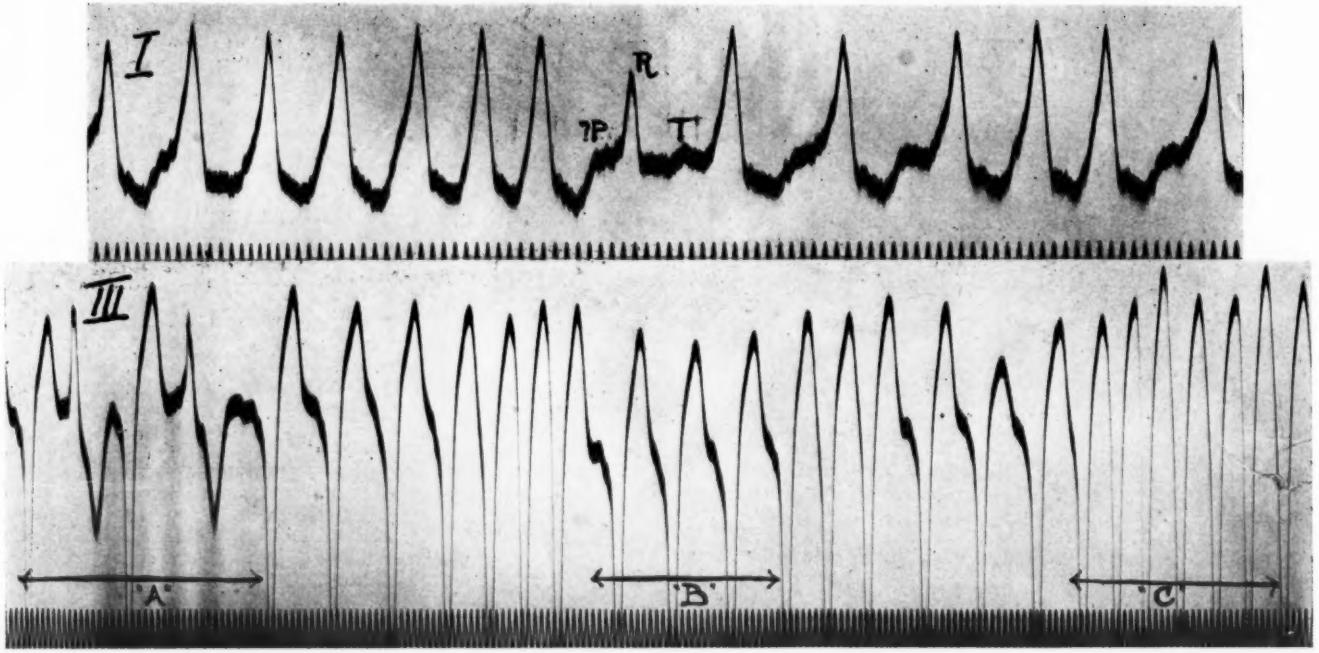

Fig. 4.—(Case 4.) These records were made on the patient's admission to hospital. The upper (lead 1) was recorded on a rapidly moving plate and shows an irregular succession of abnormal ventricular complexes. The complex marked P.R.T. is of the normal supraventricular type (compare with Fig. 5, lead 1, during normal rhythm).

The lower record (lead 3) commences with alternation in the direction of successive complexes suggesting bundle-branch defects. This is followed by a more constant type of ventricular deflection but with sudden transitions in rate. The calculated rates per minute over the periods marked *A*, *B*, *C*, are respectively 105, 190, and 320 contractions per minute. Normal rhythm is shown in Fig. 5.

pressure was applied, is demonstrated in Fig. 7. Multiple extrasystoles, with independent auricular activity, are shown in Fig. 8, and a period of normal rhythm, for comparison, in Fig. 9. The diagnosis in this case is based on the relation of the first and last abnormal complexes of the paroxysm to the normal rhythm, on their resemblance to

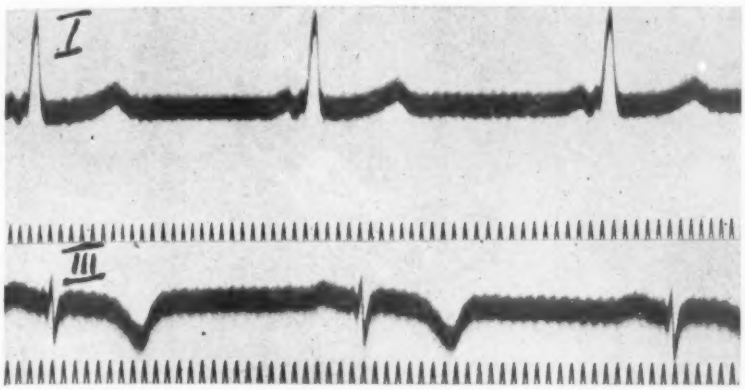

Fig. 5.—(Case 4.) These records (leads 1 and 3) were made on the cessation of the paroxysm and show the return of normal rhythm. There is marked inversion of the T-wave in lead 3. The rate is 64 per minute.

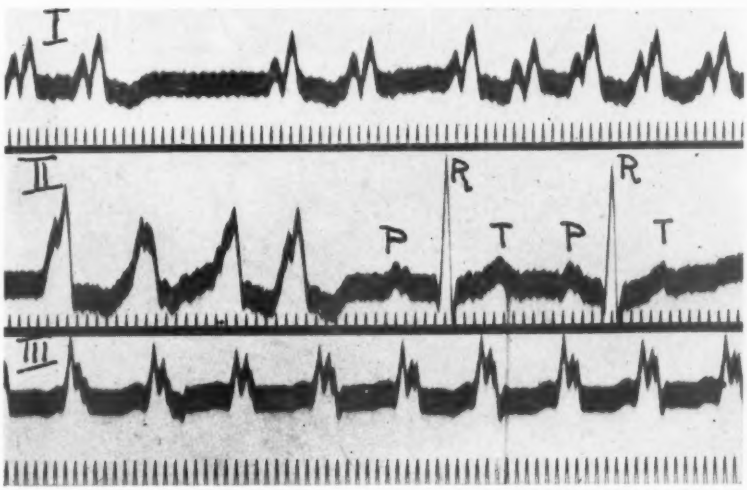

Fig. 6.—(Case 5.) These records (leads 1, 2 and 3) were taken on the patient's admission to hospital. There is a marked variation in the interventricular intervals in lead 1 (upper tracing). Lead 2 (middle tracing) shows a transient interruption of the tachycardia by two normal beats in succession and lead 3 shows the continuation of the paroxysm.

isolated ventricular extrasystoles, and at times independent rhythmic auricular activity.

DISCUSSION

These five cases appear to fall into two broad clinical groups: (1) those in advanced congestive failure under treatment with digitalis; (2)

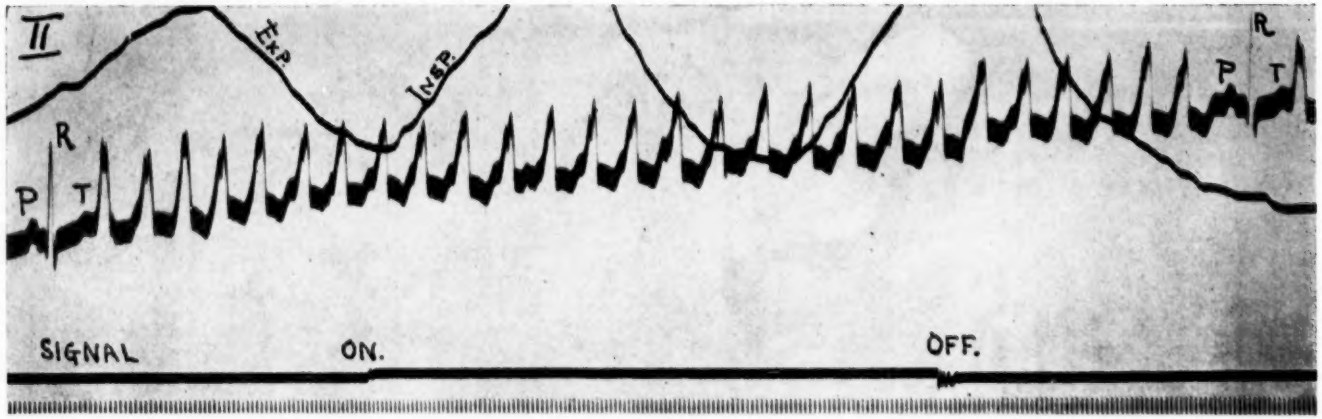

Fig. 7.—(Case 5.) Shows a short paroxysm (lead 2) of twenty-seven beats interrupting a period of normal rhythm. Bilateral ocular pressure (which slowed the heart during normal rhythm) was applied during the period marked by the signal. It was without effect. The paroxysm bore no relation to respiration (the upstroke is inspiratory). The ventricular rate is 190. Auricular activity cannot be detected during the paroxysm.

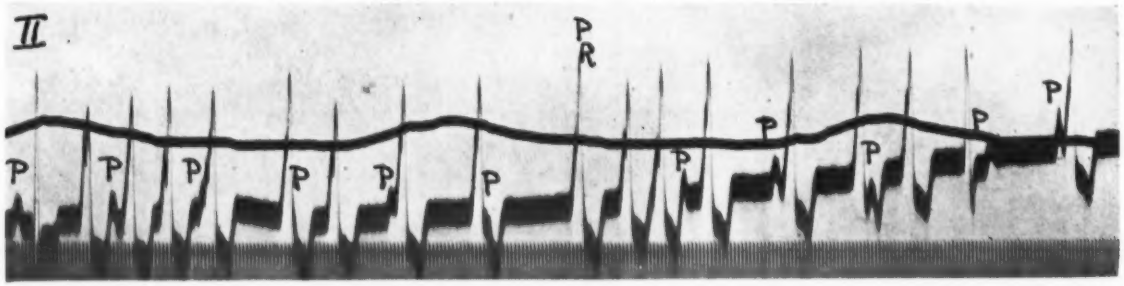

Fig. 8.—(Case 5.) This record (lead 2) commences with a normal beat and is thereafter succeeded by multiple extrasystoles irregularly interspersed. P-waves occurring regularly can be detected throughout the tracing. The premature beats bore no relation to respiration—the upstroke is inspiratory.

those in which the paroxysms, unaccompanied by congestive failure, drew attention to the state of the heart. In the first group the patients were unconscious of the rapidity of the heart's action, and complained of no symptoms before or after the attack which could be definitely attributed to the tachycardia. Congestive failure was, however, accentuated gradually as the attack proceeded. In the other group, each attack was associated with some symptom, ranging from a feeling of uneasiness to definite distress, and these patients were each conscious of the rapidity of the heart's action.

The relation of digitalis to paroxysmal ventricular tachycardia is of some interest. Though this type of rhythm is known to occur in animals after the use of toxic doses of the digitalis preparations, it is only

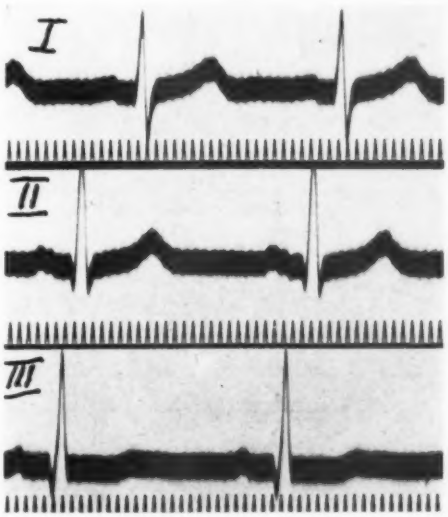

Fig. 9.—(Case 5.) This record (leads 1, 2, 3) shows normal rhythm—compare with Fig. 6.

recently that the drug has been recognized as a cause of the disorder in man. Daniélopolu[5] reported three cases of paroxysmal tachycardia following the use of digitalis and strophanthin, but it is not possible from his tracings to credit the tachycardia with a ventricular origin. Eggleston and Wyckoff[6] in their work on digitalis therapy, mention one case where recovery occurred, and Schwensen[7] has also noted the relationship. Felberbaum,[8] reporting a similar case with a regular tachycardia in which alternate complexes varied in direction, suggested digitalis as the provoking cause. Wolferth and McMillan[4] noted that in one of their cases ventricular tachycardia followed the use of digitalis, but the evidence did not suggest that the drug was the precipitating cause of the attack. Reid[9] has recently described five cases in which the amount of digitalis administered before the onset of the paroxysms

was well in excess of that indicated by the Eggleston method. In one patient in whom the ectopic tachycardia was associated with an alternating mechanism in the form of the QRST deflection, the change to ventricular fibrillation was recorded with the electrocardiograph as death ensued. These five cases suffered from an advanced degree of congestive failure associated with auricular fibrillation. Luten,[10] in his studies on the toxic rhythms of digitalis in man, noted two cases with idioventricular rhythm and abnormal complexes. In one of these, and three others,[11] an alternating mechanism was present during the paroxysms. He suggests that an advanced degree of digitalis intoxication may result in a paroxysmal tachycardia which originates at, or near, the main stem of the bundle, alternate impulses being partially

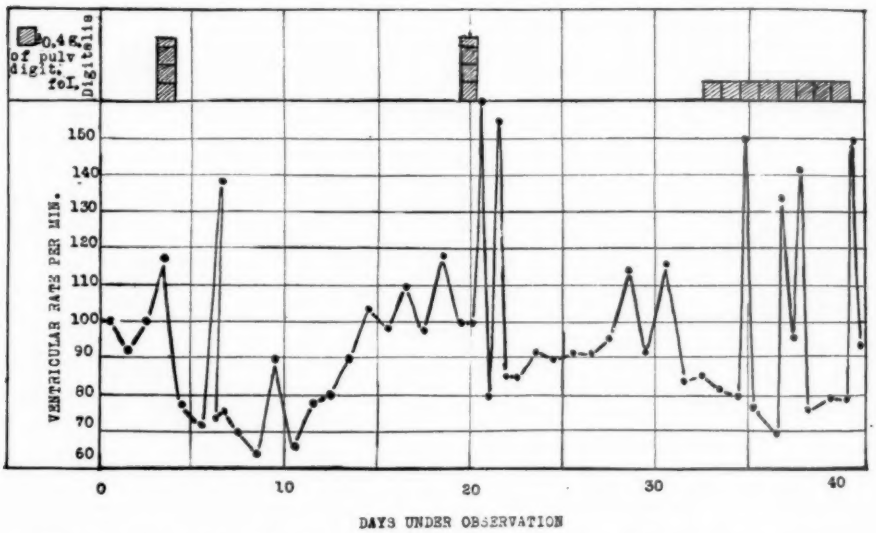

Fig. 10.—(Case 2.) This diagram shows the relation of the paroxysms of ventricular tachycardia to the digitalis dosage in Case 2. The ventricular rate charted for each day is that recorded by counting at the apex. Each massive dose consisted of 1.4 gm. of pulv. digitalis. This was calculated according to Eggleston's formula from the assumed normal weight, disregarding the excess due to ascites and edema. During the final course the patient received 0.4 gm. daily.

blocked by one branch of the bundle and transmitted by the other, and so on. This mechanism would account for the rhythmic alternation in the direction of the QRST deflection. He finds that the toxic effects of digitalis on the human heart resemble closely those observed on the heart of the cat.

Of the five cases now reported, two (Cases 1 and 2) may be definitely attributed to the use of digitalis. Case 1 received a massive dose (calculated from Eggleston's formula), of 2.8 gm. pulverized digitalis of standard potency, which was followed by slowing in ventricular rate and slight vomiting and nausea without evidence of any toxic effect on the heart. Seventeen days later a course of digitalis therapy was recom-

menced, and during this time (thirteen days) the patient received 3.4 gm. in all. Assuming a daily elimination and destruction of 0.15 gm., the amount given was therefore not in excess of that calculated by the Eggleston method. Paroxysmal ventricular tachycardia resulted, being preceded by coupled rhythm and succeeded by profuse extrasystoles and coupled rhythm, and this without general symptoms of digitalis intoxication—no nausea, anorexia or vomiting. Neither the massive dose nor the course of digitalis therapy produced improvement in the patient's state, and her progress was steady in a downward direction. The difference in the response, on the two occasions on which digitalis was exhibited in this case, might be attributed to the fact that on the second occasion the myocardium was in a more exhausted condition and more susceptible to the drug. All the cases in which this disorder of rhythm has been attributed to digitalis suffered from advanced congestive failure. An alternative explanation may be that, with the advancing failure previous to the onset of the paroxysm, the active principles of the drug were less efficiently eliminated or destroyed by the tissues.

Absorption of the drug must undoubtedly have been less rapid, and this may help to explain why the premature beats of all types persisted for upwards of ten to fourteen days after the occurrence of the paroxysm and the discontinuation of the drug. A fortnight elapsed before the full sinoauricular rhythm became firmly reestablished and, during this time, first the coupled rhythm, and later the irregular extrasystoles, gradually disappeared. A somewhat similar course of events was observed by Daniélopolu,[5] and it is probable that more protracted absorption, defective excretion and a higher degree of myocardial susceptibility towards the drug all played a part. In this case digitalis first reduced a 2 to 1 flutter to 4 to 1; it subsequently induced fibrillation. Later normal rhythm became established. A further course of digitalis was responsible for the production of paroxysmal ventricular tachycardia.

In Case 2, the subject of advanced cardiorenal and arterial disease, the relation of the paroxysms to the digitalis dosage is demonstrated in Fig. 10. In this case the paroxysms were of short duration, and, though seven in all were recorded, it was only possible to obtain electrocardiographic evidence of their nature on two separate occasions—the third and the seventh in the diagram. This patient suffered from auricular fibrillation, and the amount of digitalis administered was 0.4 gm. less than that calculated by the Eggleston method. The patient's normal weight was 126 pounds, the edema and ascites being neglected in calculating the dose. No general toxic symptoms resulted, and, save for the paroxysms, there was no other evidence of toxic effect on the heart muscle. With successive doses the paroxysms occurred earlier, until, during the final course, the first attack was noted after 1.0 gm. had been

given. This lends support to the view that those patients in whom myocardial degeneration is far advanced, and in whom the degree of heart failure is steadily advancing, become more susceptible to the drug. Apparently the underlying cause of this response to digitalis is more the state of the heart muscle than the size of the dose. In such subjects the use of massive dosage of digitalis is not without danger.

Cushny,[12] in an attempt to reconcile experimental and clinical results in the use of digitalis, has put forward the view that in clinical auricular fibrillation the characteristic reaction to digitalis depends on the state of nutrition of the heart, as well as on the presence of the fibrillation, malnutrition and a high ventricular rate favoring a good response to digitalis. Nutrition must, for the present, be regarded as a relative term, for a time must come when, with advancing failure and increasing malnutrition, the ventricle is unable to avail itself of the proffered help. Nutrition of the heart muscle is an important factor in determining the heart's response.

Concerning the production of paroxysmal ventricular tachycardia by digitalis, it is of interest to note that in cases of myocardial failure with normal rhythm, digitalis may induce auricular fibrillation. This has been observed in man by Mackenzie,[13] Daniélopolu,[14] and others. It has also been noted by the writer in four cases, after full massive dosage with digitalis,[15] and Resnik[16] has recently investigated this subject in seven patients showing a high degree of myocardial failure, who developed transient auricular fibrillation after digitalis therapy. He concludes that the probable predisposing factor is myocardial failure, and that digitalis acts mainly on the muscle and possibly also through the vagus in inducing this change in rhythm. May it not be that some similar mechanism underlies the production of transient paroxysmal ventricular tachycardia after the use of digitalis? Lewis' views that auricular fibrillation is a manifestation of circus movement in the auricle are now widely accepted, and the suggestion has been put forward that a similar mechanism is responsible for ectopic tachycardia in the ventricle. Circus movement, as applied to the auricle, and ventricular tachycardia, have certain features in common (Lewis[28]), and it seems probable that, given a certain critical state of the heart muscle, e.g., profound malnutrition, either of these disorders may be provoked by digitalis. The manner in which digitalis induces ventricular tachycardia is not fully understood, but Robinson and Hermann[17] observed ventricular paroxysms following coronary obstruction in man, and there is evidence to suggest that large doses of digitalis may constrict the coronary arteries, at least under experimental conditions.[18] In the present state of our knowledge, any discussion on the mechanism of the action of digitalis on the ventricular musculature, embarrassed by such a complex process as advancing failure, must needs be purely specula-

tive. The drug increases rhythmicity,[10] produces extrasystoles, depresses intraventricular conduction, and it is conceivable that, in the presence of a certain critical state of the heart muscle, the refractory period may shorten. Such a combination would favor the establishment of circus movement. Leaving aside theoretical considerations, the important point in the production of abnormal rhythms by digitalis appears to be the state of nutrition of the heart muscle.

The remaining three cases may now be briefly discussed.

Case 3 was the subject of advanced arteriosclerosis, and during the attack, which was only of about one hour's duration, the patient complained of no symptoms except a vague uneasiness and fluttering in the chest. The attack occurred twenty-four hours after commencing a course of iodides. The patient was not aware of previous paroxysms. He collapsed and died suddenly in the street one week after leaving hospital. This case illustrates a different type of mechanism from the others in this series, in that the rate of the auricle was precisely half that of the ventricle, alternate impulses being retrograde to the ventricle. Similar curves of reversed heart-block have been published by Hart.[30] The precipitating factor in this case could not be determined, but there is little doubt that the underlying cause was coronary sclerosis and myocardial degeneration.

Case 4, in whom the abnormal rhythm was regarded as a manifestation of nicotine intoxication, is included in this series on account of the unusual curves obtained during the paroxysm. It is admitted that the evidence for a ventricular origin of the paroxysm is not complete, and that the necessary criteria cannot be presented in support of such a conclusion. We have no records of the beginning or end of a paroxysm; isolated extrasystoles, though present, were not recorded graphically, and auricular activity is undefined during the attack. In Fig. 4 the complexes are inconstant in form and the rate varies greatly.

The record opens with alternation in the direction of successive complexes and later becomes characteristic of left ventricular extrasystoles. This may be attributed to either a supraventricular focus with alternate conduction defects in the bundle branches, or to an active focus of impulse production in each ventricle. The final beat of this rhythm is followed by a period of diastole of greater duration than any other present in this record and during which it might be assumed that the impulse center changes its location. The succeeding complexes now become of a more uniform type and assume the form characteristic of left ventricular extrasystoles. To explain the alteration in the rhythm, it may be assumed that either the site of impulse formation is shifted from above the bifurcation of the bundle to the left ventricle in the one case, or in the other, that the excitable center in the right ventricle

becomes overwhelmed by the increasing activity of that in the left. In the case reported by Castellano[19] the complexes varied in type and the ventricular rate ranged from 150 to 240 during the paroxysms. Great fluctuations in ventricular rate are clearly visible in the records of this patient. In Fig. 4 (lead 3) the rate of the last six complexes is 320 per minute, and of the three beats in the middle of the same record 190 per minute. During the period of alternation in the direction of successive complexes the rate is 105. During the highest rates of beating, the complexes are of uniform type, and succeed each other so rapidly that a fresh complex commences before the completion of the preceding *T* wave. Such sudden transitions in rate are unusual in paroxysmal tachycardia, though Marvin[20] observed a gradual slowing in rate over a period of five days in a case of paroxysmal ventricular tachycardia. Feil and Gilder[21] noted in the auricular type that the complexes were of great regularity, the average variation between successive complexes never exceeding 0.01 sec., but dealing with ventricular paroxysms, Strong and Levine[22] found that the variation might be ten to twenty times as great. The maximum variation in several records was 0.191 sec. in the case under discussion.

In Fig. 4 (lead 1) the eighth complex is of a distinctly different type from the others and resembles very closely the normal complex (Fig. 5) of the same lead. The *P* wave of this beat is poorly defined, but the deflection is of the supraventricular type. On account of the fluctuation in rate and the abnormal form of the complexes the paroxysm is assumed to be of the ventricular type. Against this is the known fact that, given a high rate of impulse production the ventricular deflections may often be anomalous, as pointed out by Lewis. Whether or not the paroxysm be of ventricular origin, the excitation wave undoubtedly pursues an abnormal course through the ventricular muscle.

In considering the action of nicotine on the human heart it may be of interest to refer to the experimental work of Clerc and Pezzi.[23] In their studies on the response of the dog's heart to intravenous dosage with nicotine, they observed various disorders of rhythm, which they divided into two stages: (*a*) a preliminary bradycardia of vagal origin, followed by (*b*) a period of tachycardia. The latter was accompanied by auriculoventricular dissociation and an irregular ectopic tachycardia of ventricular origin. Nicotine acts on the ganglia of the autonomic nervous system, first stimulating and later paralyzing them, and according to Wertheimer,[24] it has also a direct action on the heart muscle. It would seem probable that in this patient nicotine was at least a factor in the production of the paroxysms. The man suffered from a secondary anemia, and this, by embarrassing the nutrition of the myocardium, may have tended to augment the toxic effect of the drug on the heart muscle.

Case 5 is an example of this disorder of rhythm occurring in an apparently healthy young man. No abnormal signs could be discovered on physical examination, and it was the repeated paroxysms which drew attention to the state of the heart. Similar cases have been reported by Scott[25] and Gallavardin.[26] For eighteen months this patient was experiencing from four to six attacks a day and, previous to his admission to hospital, their severity was increasing. The more severe attacks were succeeded by profuse extrasystoles arising from a constant focus, and by many short paroxysms lasting from a few seconds to several minutes. Multiple extrasystoles were numerous for three or four days after a severe attack. They gradually became less frequent from day to day as the normal rhythm daily became more firmly reestablished. Isolated extrasystoles were frequent from time to time, and simple exercise tests constantly provoked them, or brief paroxysms, even when under full digitalis dosage. Vagal stimulation, effective when the heart was beating in normal rhythm, failed to abolish the paroxysms. Rest in bed and digitalis reduced the frequency but failed to abolish completely the attacks, and though the patient's condition improved, any muscular activity provoked extrasystoles which, if continued, readily induced paroxysms. Digitalis in sufficient dosage to cause anorexia and vomiting, did not, under resting conditions, provoke paroxysms. This lends support to the view that in advanced congestive failure the mechanism of the drug's action on the ventricle is altered. This patient had no congestive failure, and there was no evidence that full dosage with digitalis was in any way harmful.

Quinidine was definitely of benefit, in that the tendency to extrasystoles was greatly diminished, and that the patient was able to undertake more strenuous exertion with than without it. The optimum dose was found to be 7 grains per day, and in time it is hoped to reduce this dose gradually. The patient has had no attacks since commencing this line of treatment, and it has enabled him to return to work. When quinidine is discontinued multiple extrasystoles reappear. Scott[25] found quinidine of value and suggested that it prolonged the refractory period of the ventricle, which view has recently been confirmed by Drury.[29]

Of the five patients, two died while under observation, forty-three days after the occurrence of the paroxysm in Case 1, and thirty-six days after the first paroxysm was noted in Case 2. In Case 3 death took place suddenly eleven days after the attack. In Case 4 the patient was able to return to his work, but recent efforts to trace him have been unsuccessful. In Case 5, with no demonstrable cardiac lesion, two years have now elapsed since the first attack. There are conclusions to be drawn which, even from such a small number of cases, may be helpful in forming a prognosis.

In the presence of a demonstrable cardiac lesion with advancing fail-

ure and under treatment with digitalis, the occurrence of this form of tachycardia is of the most serious significance. It indicates that the ventricle is incapable of responding to the drug in a satisfactory manner, and, apart from the danger of ventricular fibrillation, this disorder places an additional burden on the heart and gradually embarrasses all the bodily functions. Such cases call for special care in the use of digitalis. Unless accurate observations are made by closely watching the heart rate, attacks may pass undetected, as in these subjects they are often unassociated with suggestive symptoms and are accompanied by a large pulse deficit. Congestive failure, increasing in amount during the course of a few hours, points to the presence of a change in the type of rhythm, and requires careful investigation. In the absence of advanced heart disease, the prognosis must be based on the underlying state of the myocardium, the frequency and duration of the attacks, and more particularly, on the patient's reaction to this type of rhythm and the degree of failure induced. Finally, a good response to treatment and the possibility of eradicating any suspected or exciting causes render the outlook more favorable. As a general rule, each paroxysm must run its own course. In these five cases the usually recommended procedures, such as vagus stimulation, abdominal pressure and deep breathing, were all ineffective in each individual. Quinidine controlled the attacks in Case 5, but in the presence of advanced myocardial disease, the use of the drug is not warranted. Calcium chloride intravenously appeared to be of some value in the case reported by Orsi.[27]

SUMMARY

Paroxysmal ventricular tachycardia is more common than the number of reported cases would indicate. Five cases are described, illustrating different causes for the attacks.

In two, the subjects of advanced myocardial disease with steadily progressing congestive failure, digitalis was the provoking cause of the attacks. The dose of digitalis did not exceed that calculated by the body-weight method of Eggleston. It is concluded that this disorder of rhythm depends more upon the state of cardiac nutrition than upon the degree of concentration of the drug in the tissues. It is suggested that a mechanism, similar to that which underlies the production of auricular fibrillation by digitalis in cases of heart disease with normal rhythm, may also be present in these cases where the drug has provoked ventricular tachycardia.

In one case, nicotine intoxication was regarded as an important factor in the production of this arhythmia.

Advanced coronary sclerosis and myocardial disease were present in one case, and in the last case no demonstrable cause could be found.

Quinidine sulphate was of value in controlling the paroxysms in this case.

The prognosis is briefly discussed and emphasis is placed upon closely watching the heart rate in all cases of advanced myocardial failure under treatment with digitalis.

My best thanks are due to Dr. Robert A. Fleming, for permission to report Case 4; to Professor Edwin Bramwell, for Case 5; and to Professor D. Murray Lyon, for the facilities he has given to make observations on the remaining cases of this series; to the Trustees of the Clinical Medicine Research Laboratory, for permission to work there; and to Mr. R. Danskin, technical assistant.

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PAROXYSMS OF TACHYCARDIA OCCURRING IN A CASE OF HODGKIN'S DISEASE IN WHICH THE VAGUS NERVES WERE DEGENERATED BY THE PRESSURE OF ENLARGED MEDIASTINAL GLANDS*

VERNE C. GRABER, M.D.
IOWA CITY, IOWA

IT IS believed that the interest in the relationship of the extrinsic cardiac nerves to paroxysmal tachycardia will justify a brief report of the following case:

W. E. P., aged thirty-five years, entered the University Hospital April 30, 1924, complaining of a feeling of fullness in his chest, cough, temperature, loss of weight, and weakness. His past medical history was negative except for a mild attack of influenza in 1918 and a pneumonia in 1923. There was no history of diseases of the rheumatic cycle and he denied venereal infections. He was married and his wife and one child were alive and well. There had been no miscarriages.

His illness began in February, 1924, with what was thought to be measles. He was mildly ill for about one week. Following this his appetite was poor and he did not regain his strength. Subsequently a feeling of fullness appeared in his chest. This was associated with a dull, boring sensation. He also had an irritating, non-productive cough and observed that he had a temperature of 100° F. in the evening. He was examined by a physician and told that he had some trouble in his lung. Within a few days the patient discovered small glands in the right side of the neck. A second physician was consulted who stated that the glandular enlargement was similar to those seen in Hodgkin's disease. X-rays of the chest, and blood counts were, however, not made. The patient denied symptoms referable to the heart. He estimated that he had lost twenty pounds in weight and seventy-five per cent of his strength. He had not been able to work since the onset of his illness.

Physical examination.—The weight in street clothes was 126¾ pounds. His normal weight was 146. The temperature was 100° F. at 4:00 P.M. He was tall, slender, sallow and noticeably emaciated. The voice was a little husky. The tonsils were atrophied. The tongue was pale, flabby and slightly coated. The neck appeared full. On the right side a group of large, soft, discrete lymph nodes, which were not adherent to the skin, was felt. A corresponding group was felt in the right axilla. The ileo-inguinal nodes were a little larger than normal. The thorax was symmetrical. There was no retrosternal dullness. The lungs were normal, except for a little prolongation of expiration at the right apex above and below the clavicle. A few persistent crackles were heard at the right supraspinous fossa. Whispered voice was normally transmitted.

The heart was normal in size. The sounds were distinct and regular. There were no murmurs. The rate was 104 per minute. The pulse wave was rather small, but otherwise satisfactory. The systolic blood pressure was 110 mm. and the diastolic 60.

In the examination of the abdomen a deep-lying resistance was felt over the area between the xiphoid process and the umbilicus and laterally to rectus muscle. No masses, however, were palpable. The liver and spleen were not felt. There was no edema of the shins. The neurological examination was negative.

*From the department of Internal Medicine, State University of Iowa.

Laboratory findings.—The hemoglobin was 52 per cent; red blood cell count 3,870,000 and white blood cell count 18,800. In the differential count of 100 cells 88 per cent were polymorphonuclears and 12 per cent small lymphocytes. The red blood cells were pale and varied in size and shape. They, however, stained satisfactorily. The blood platelets were estimated to be 625,000 per c.c. Throughout the course the blood remained practically the same except for a gradual increase in the leucocyte count to 30,800.

The urine was essentially negative throughout the course. The sputum contained varying amounts of blood. Repeated examinations for tubercle bacilli were negative. The blood Wassermann was negative. The x-ray plates of the chest showed a shadow suggestive of enlarged mediastinal glands. There was, however, no increase in the lung density, and the cardiac and diaphragm shadows were normal.

Clinical course.—During the hospital observation there was no apparent change in his condition until two days following admission. At this time he noted a sud-

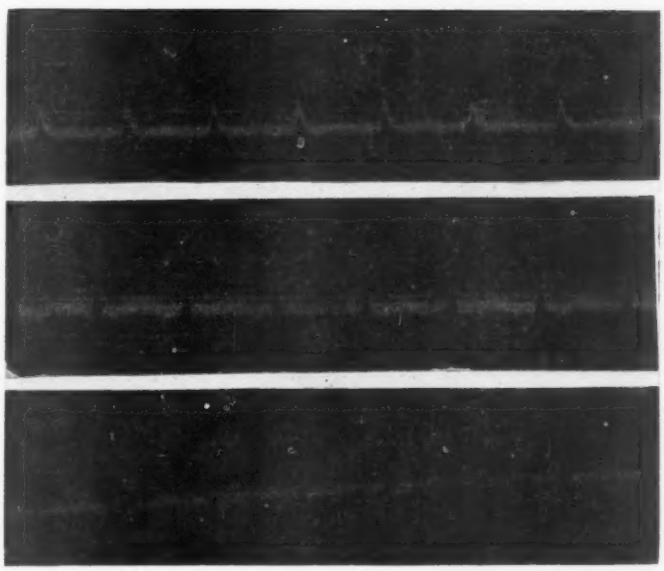

Fig. 1.—This electrocardiogram was taken during the paroxysm of rapid heart action. The P-wave follows the R deflection. The cardiac rate was estimated to be approximately 180 per minute.

den onset of rapid heart action. Three hours later an electrocardiogram was taken which showed a paroxysmal tachycardia of the auriculoventricular type. The cardiac rate was approximately 180 per minute. This attack persisted for eight days. The only evident signs of cardiac embarrassment were slight dyspnea and a little cyanosis of the lips and finger tips. The attack ended abruptly, and a cardiac rate of 96 per minute was resumed. The electrocardiogram showed a normal cardiac mechanism.

Two days following the cessation of the tachycardia the patient was allowed up in a chair for one-half hour. Shortly after returning to bed he again became conscious of a rapid heart action. The electrocardiogram again showed a cardiac mechanism similar to that of the previous attack. This paroxysm lasted about twenty-four hours. The following day the tachycardia returned and persisted until death. Vagal pressure and injections of atropine, gr. $\frac{1}{150}$ produced no change.

On the eighth day following the onset of the final attack of rapid heart action,

the area of cardiac dullness was noted to be definitely increased in size; the signs of fluid in the right chest were discovered; the liver extended 12 cm. below the costal margin and edema appeared in the lower extremities. The course was progressively downward and the patient died eleven days later from cardiac failure.

Necropsy.—In this report only the necropsy findings relative to the cardiovascular system and those of particular interest concerning the Hodgkin's disease will be given.

The body was well developed and slightly undernourished; the ears, neck and lips were cyanosed; the feet, shins, dependent portions of the thighs, scrotum and penis were markedly edematous. The veins of the neck were prominent. The veins of the forearm and temples were full and distended. There was a mass of lymph nodes above the right clavicle. Other nodes were felt in the deep and posterior triangle of the neck and under the left clavicle. The nodes of the axilla and groin were

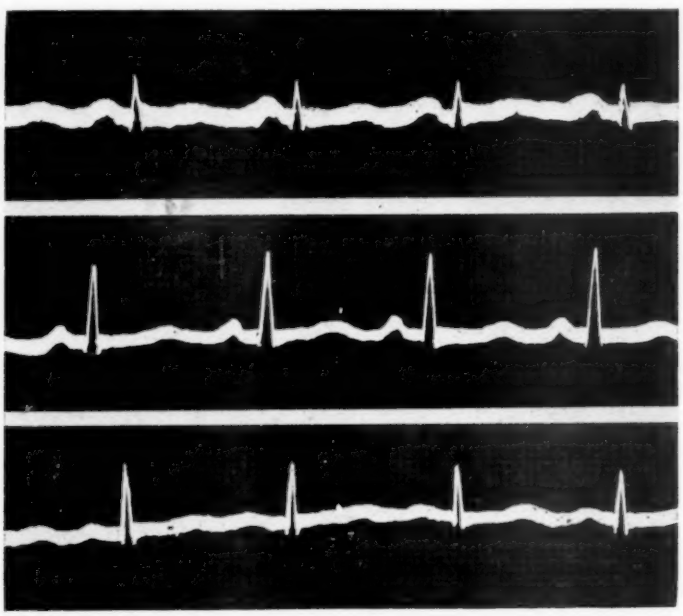

Fig. 2.—This represents a reconstruction of the electrocardiogram taken during a period of normal cardiac mechanism following the first paroxysm of tachycardia.

definitely enlarged. The abdomen was distended. Six hundred c.c. of clear, straw-colored fluid was removed from the peritoneal cavity. The mesenteric lymph nodes were large; they measured from ½ to 2 cm. in diameter. The right pleural cavity contained 2,100 c.c. and the left contained 200 c.c. of blood-tinged fluid.

The nodes of the anterior and posterior mediastinum along the trachea and at the hilum of the lungs were enlarged. A large mass of nodes was also found between the aorta and the trachea. The main trunks and branches of the right and left vagus were markedly flattened out by pressure in the pulmonary plexus and anteriorly to the trachea.

The pericardial cavity contained 60 c.c. of straw-colored fluid. Both visceral and parietal layers were smooth.

The heart weighed 310 grams. It was distinctly pale in color. The right auricle was very flabby, and the consistency of the left ventricle was definitely decreased. The valves were normal. No gross infiltration of the heart could be made out.

A few nodules were found in the lungs and spleen.

The liver weighed 1850 gm. It extended a hand's breadth below the costal margin. It showed marked passive congestion, but no evidence of infiltration.

The bone marrow was intensely red in color.

Microscopical findings.—The lymph nodes showed the characteristic architecture and accessory findings of Hodgkin's disease. The muscle fibers of the heart were swollen and in places definitely hydropic. There was some fragmentation and a slight amount of brown atrophy. The vessels were normal.

Sections from the right and left vagus nerve, after leaving the pulmonary plexus and sections of the branches of these nerves to the deep cardiac plexus were examined by March's method and in each instance the nerve tissue was found to be largely replaced by fat.

DISCUSSION

The clinical course, as confirmed by the autopsy findings was one of a rapidly progressing case of Hodgkin's disease. The onset of the paroxysmal tachycardia led us to feel that it might be the result of direct action of the enlarged mediastinal glands on the extrinsic nerves of the heart, either by pressure or edema. At necropsy the vagus nerves were found to be extensively damaged by pressure.

There is no definite evidence, to our knowledge, that the action of the extrinsic cardiac nerves may be primarily responsible for the production of paroxysmal tachycardia, yet isolated instances have been reported in which the condition was apparently associated with derangement of those nerves. Galli[1] was able to induce paroxysmal tachycardia in his patient by the hypodermatic injection of atropine. Kure[2] cites a child in whom attacks were provoked by the doing of problems in arithmetic. There are also a few instances in which the onset of paroxysmal auricular fibrillation appeared to be related to the action of vagus and sympathetic nerves. Smith and Moody[3] have recently reported a case in which an attack of absolute irregularity was induced by deep breathing. This was more easily accomplished when the patient was under increased nervous tension or following the administration of epinephrin. These investigators had another patient in whom auricular fibrillation appeared subsequent to the injection of epinephrin. They were inclined to believe that while the onset of the abnormal cardiac mechanism in the first patient was provoked by vagal action, it was also influenced by the tone of the sympathetics. In the second patient the influence of the sympathetic nerves was even more marked. In this case premature contractions were regularly induced by forced breathing prior to the administration of the epinephrin. The findings in these two cases would seem to be comparable to the observations of Levy,[4] who has shown that the stimulation of the sympathetics has the same effect as section of the vagi.

It is to be recalled that our patient did not admit to having had previous attacks of paroxysmal tachycardia. Furthermore there was noth-

ing in his past history or in the physical findings suggestive of cardiovascular disease at the time of the first examination. It is therefore believed that the destruction of the vagus nerve influenced the production of the abnormal cardiac mechanism.

We wish to express our thanks to Dr. C. P. Howard for permission to publish this case report, and also to Dr. G. H. Hansmann for his careful study of the tissues.

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GAIN IN WEIGHT AS A GUIDE IN THE CONVALESCENT CARE OF CARDIAC CHILDREN*

HUGH MCCULLOCH, M.D.
ST. LOUIS, MO.

THE ideas expressed in this paper are based on observations on children with heart disease who have been at Ridge Farm, the country convalescent department of the St. Louis Children's Hospital, and in the Cardiac Clinic of the Washington University Dispensary during the past six years. A direct continuity of service exists between the clinic, hospital and sanitarium care, so that observations on the same child extend through various stages of the disease. Children with illness other than heart diseases are admitted to Ridge Farm, though in all, the essential problems are undernutrition and chronic infection. The long period of convalescence from rheumatic fever and heart disease, the importance of proper care during this period to avoid a recurrence of attacks and to insure a favorable outcome, and the lack of more definite signs of the progress of the disease make us feel that any criterion by which one can gauge this progress will be of value to the physician in his handling of such children.

That there is some relation between body nutrition and resistance to infection hardly need be discussed. It is a matter of common knowledge that good nutrition is associated with increased resistance to infection, though other factors play a part in establishing resistance. An example of the good results which come from improved body nutrition and from building up what has been termed a "nonspecific immunity" has been shown by a study of the prevention and control of tuberculosis.

If one accepts the gain in weight as a guide in the care of cardiac and other types of malnourished children, this gain must be considered in relation to the normal body weight and use must be made of various criteria for estimating this normal weight. Without entering into a discussion of the relative values of the various methods of estimating nutrition, height-weight-age tables offer a means for comparing the individual child with averages of large groups of children. It is also essential to have some knowledge of the physical condition of the child's body as shown by examination of the skin, subcutaneous fat and muscles.

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*Read before the American Heart Association, Dallas, Texas, April 20, 1926.
From the Department of Pediatrics, Washington University School of Medicine and the St. Louis Children's Hospital.

A gain in body weight does not necessarily indicate an improved body nutrition and an increased resistance to infection, yet in children this is usually true. The undernourished child should show a rapid gain when intensive nutritional care is instituted. Older children, especially, show a quick response. As the individual normal weight is approached, the rate becomes slower until the periodic increase corresponds to that shown by normal children. The rate of gain may be of some importance, since the rapid rate shown by the undernourished child might, in a child of normal body weight, indicate some altered nutrition.

It must be assumed that the heart muscle shares in the nutrition of the body in the same way as skeletal muscle. Direct evidence of this fact is lacking, though it is a matter of frequent observation that, in the anemias and other conditions with low general or visceral muscle tone, heart weakness may be associated with signs of fatigue. While the physiological phenomena of cardiac muscle function are different in many respects from other muscle tissues, it is probable that the metabolic processes are similar. Experimental evidence supports this view.

It is agreed that when an active heart infection is first recognized in a child, absolute rest in bed is necessary to prevent further damage to the heart. When, after a time, it seems advisable to allow the child more activity, the relationship of weight and nutrition may be watched, in connection with the leucocyte count, pulse rate, fever or other symptoms, as a guide in the progress of the convalescence. During the period of rest in bed there is frequently noted a gain in weight similar to that of tuberculous individuals while resting in bed.

After the period of activity of the infection in the heart has passed, and when convalescence is established, exercise may be allowed the child in increasing amounts so long as the weekly or daily weight is increasing. Our custom at Ridge Farm is to allow a child an increase in exercise after he has gained the expected amount the previous week. If he has not gained he is not allowed increase in exercise, and if he has lost weight he is either put back to bed or the allowance of exercise is decreased.

The signs which cardiac children frequently show preceding a recurrence of the rheumatism, the heart infection or chorea, are of importance and should be noted in connection with the periodic body weight. There are four signs which can be noted by the physician, visiting nurse or by parents after sufficient instruction. If repeated observations show one or more of these signs to be consistently present, an impending flare-up of the infection may be anticipated:

1. A resting pulse rate increasing from the basal rate to 100 or more per minute.

2. A daily variation in the body temperature of more than 1° F., particularly if the rise be to 100° F. or above. This point is of importance whether the rise of temperature be caused by infection in the heart or elsewhere in the body.

3. A lack of appetite, loss of sleep and general inertia.

4. A relative or an absolute increase in the neutrophilic blood cells.

Two important events may take place in children who are convalescing from active heart disease. The focus of infection from which the disease originated may be still active or show periods of activity, in which event the child may fail to gain in weight or

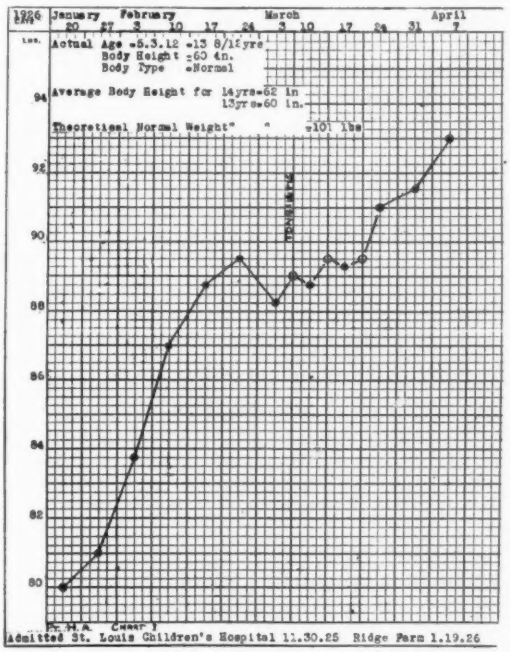

Chart I.

actually lose until the focus can be cleaned up. This applies especially to infected tonsils and sinuses. (See Chart I.)

Secondly, the attacks of rheumatism or activity in the heart may show a tendency to recur. When these relapses or recurrences take place they are usually preceded by a period of loss of weight extending over a week or more. The child should then be restricted as to exercise or kept in bed in an effort to prevent a serious relapse. Such procedures may not avert the recurrence, since proliferative changes in body tissues, due to rheumatism, may be present without exudative signs. It is true that attacks and recurrences take place, even while the child is continuously at rest and under the influence of salicylates. (See Chart II. Chart III.)

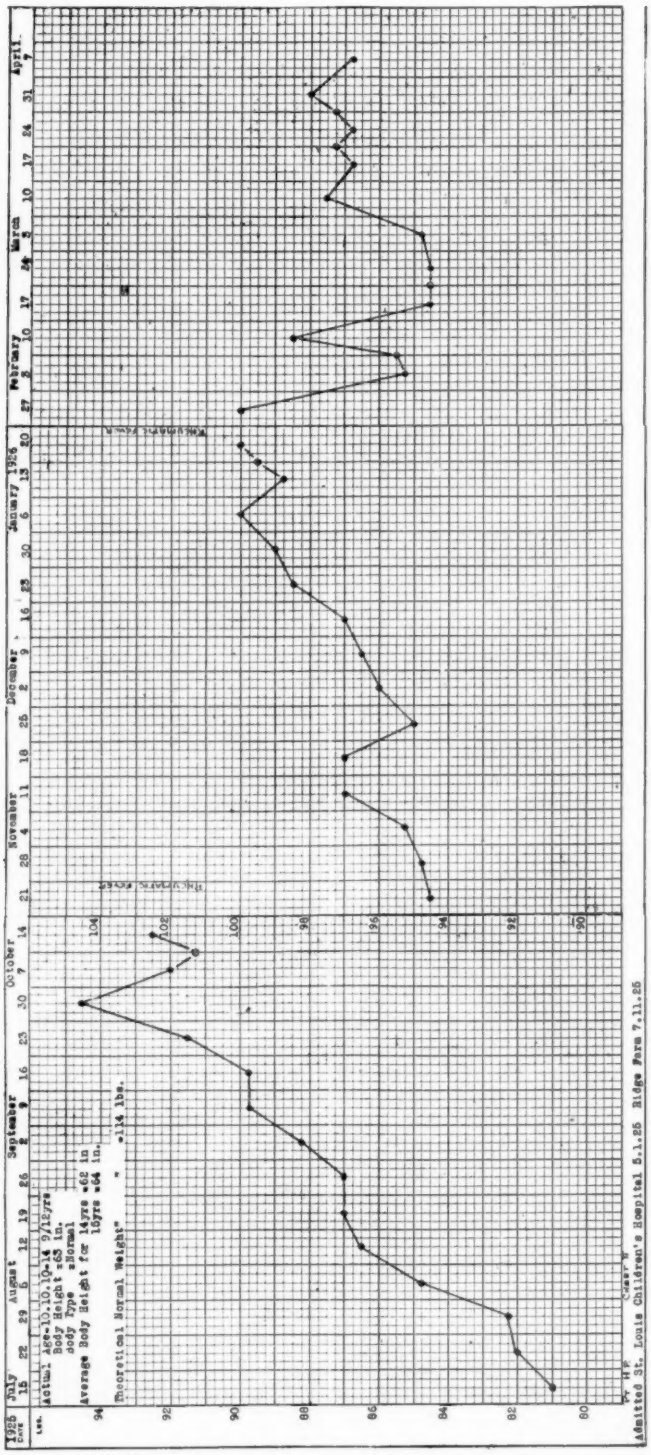
1925
July
August
September
October
November
December
January 1926
February
March
April
Actual Age-13.10.10-14 9/12yrs
Average Body Height for 14yrs =62 in.
Theoretical Normal Weight" =114 lbs.
Admitted St. Louis Children's Hospital 5.1.25 Ridge Farm 7.11.25

Chart II.

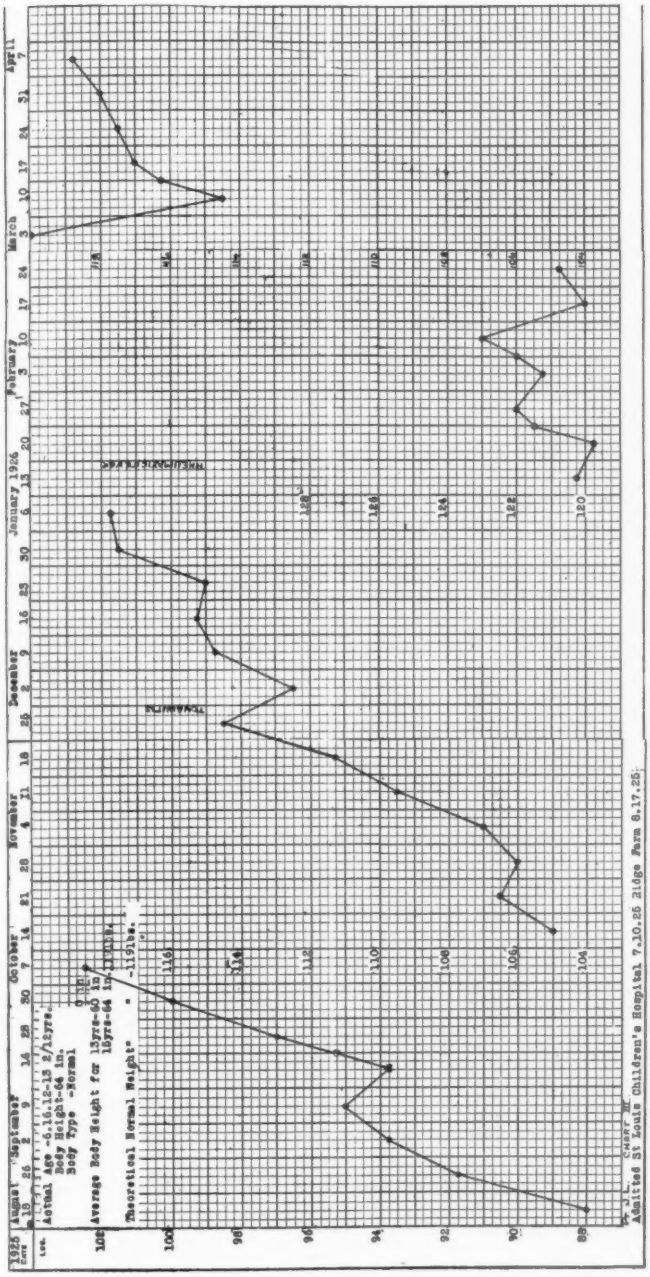
1925
August
September
October
November
December
January 1926
February
March
April
Admitted St Louis Children's Hospital 7.10.25 Discharged Farm 8.17.25.
CHART III
Average Body Height for 13yrs-60 lbs
Theoretical Normal Height
Actual Weight

Chart III.

The presence of infection in the body of a child constitutes a physical defect which hinders gain in weight. This is true whether the infection be in the heart, in the tonsils or in some other structure. This fact has been pointed out by Emerson[1] and by Kaiser[2] and is frequently mentioned in the voluminous literature on focal infections. Until the area of infection becomes quiescent or can be cleared up, the child cannot be free to gain. This raises the important question of the time to clean out areas of excessive infection in the tonsils, nose or paranasal sinuses. Our opinion in regard to this question is that no operative procedures should be undertaken so long as the child is gaining weight and improving. If relapses or rheumatism occur and if with proper rest in bed and care there is no gain in weight, it may be necessary to assume the risk and prematurely remove tonsils and clean out sinuses. The safer procedure is to allow the child to gain up to normal weight and to full exercise and then to operate. We have had the frequent experience of seeing a child gain up to a certain point, after which time no further increase took place until the tonsils were removed.

Congenital heart disease may be of such a nature as to interfere with proper growth and nutrition. This may or may not be related to the degree of cyanosis, but it has been our experience that these children do not respond to the usual care, and even under most favorable conditions fail to show a proper growth. It is not clear how some of these heart lesions can so seriously interfere with growth in such children, nor is it clear why other children with apparently similar defects grow in a normal manner.

The measures which should be instituted to improve the nutrition of these convalescent cardiac children are similar to those used in the sanitarium care of convalescent tuberculous patients. Briefly, these measures are: (1) physical and mental rest, (2) attention to diet and food habits both as to quality and quantity, (3) heliotherapy and (4) removal of foci of infection. Our study of the care of these children impresses us with the great value of sanitaria as an opportunity for using heliotherapy. Its value to these children is no less than to the tuberculous patient.

The importance of gain in weight is greatest during the period of malnutrition and convalescent care of heart disease, but it is still useful as a guide when the weight is normal. When the heart disease is inactive, or when the child has been returned to school and a normal routine, the various points outlined above all hold true. The periods of observation may be not so frequent, though the same signs must be looked for. Children returning to the Cardiac Clinic during the subsequent months or years after the heart disease has become

arrested should maintain a normal body weight, show seasonal gain in weight, show a growth in height and remain free of symptoms.

CONCLUSIONS

1. Cardiac children usually show some degree of malnutrition as a result of, or associated with, the infection which causes the heart disease.

2. Gain in weight is a sign of favorable progress of the convalescence. The weight should, therefore, be recorded regularly in order to anticipate relapses of the rheumatism or heart disease.

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(*For discussion see page* 649.)

CLINICAL FEATURES OF CASES EXHIBITING ELECTROCARDIOGRAMS CONFORMING TO THOSE OF EXPERIMENTAL COMPLETE BUNDLE-BRANCH BLOCK*

FREDRICK A. WILLIUS, M.D.
ROCHESTER, MINNESOTA

DURING the last decade numerous and varied opinions have been published, dealing with abnormalities of the initial ventricular complex (QRS) of the electrocardiogram. However, opinions regarding these graphic changes are becoming more and more crystallized, owing to numerous experimental studies and to the accumulation and correlation of larger groups of clinical cases.

Lewis and his coworkers have shown clearly that the excitation wave follows a definite course in its spread over the ventricular muscle, entering the ventricles by way of the His bundle, passing along the two main branches of the bundle and their ramifications and spreading through the Purkinje plexus which lines the inner surface of both chambers. The excitation wave spreads outward from the Purkinje structure through the ventricular walls toward the outer surface of the heart. The excitation wave travels ten times more rapidly in the special conduction system than in the ventricular musculature proper.

By a careful recalculation of Lewis' original experimental curves Wedd and Stroud showed that when a correction was made for its full development, the period of spread of the excitation wave in the ventricle corresponded to the duration of the QRS complex.

The *form* of the QRS complex is then dependent on the course of the excitation wave through the ventricular musculature, and the *duration* of the QRS complex, on the time consumed by the excitation wave in completing its course.

In 1910 Eppinger and Rothberger reported their experimental work on dogs in which they severed the main right and left branches of the His bundle by introducing a small knife through the wall of the ventricle. When the right branch was cut, the normal ventricular complex was replaced by a diphasic complex of high amplitude. The QRS complex was directed downward, its duration was increased, and the T-wave was directed upward and exaggerated in a single lead from the esophagus and the rectum. Division of the left branch revealed similar changes except that there was reversal in the direction of the complexes. In the same year Eppinger and Stoerk recorded electrocardiograms of two cases coming to necropsy in which lesions were demonstrated which completely divided the right bundle branch. The electrocardiograms re-

*From the Section on Cardiology, Mayo Clinic.

vealed diphasic QRS complexes in all leads, with increased amplitude in Leads I and III and of greater duration than normal. The QRS complexes were directed upward in Lead I and downward in Leads II and III.

Rothberger and Winterberg repeated this experimental work, using the three usual leads, and in 1913 published their observations. After division of one of the main bundle branches they obtained typical diphasic complexes, but the QRS complexes in Leads I and III led in the same and not the opposite direction, as in the cases reported by Eppinger and Stoerk.

Lewis again investigated the subject in 1916, conducting his experiments on dogs. After division of the right bundle branch he found, in most instances, that the QRS complexes were directed downward in all leads. Lewis called these electrocardiograms "concordant". In a few animals the QRS complexes were directed upward in Lead I, and these tracings were called "discordant". The fact that right bundle-branch block in man produced discordant electrocardiograms, and in most dogs concordant curves, was an apparent incongruity, but Lewis showed that it resulted from anatomical differences in the hearts. The hearts which produced discordant curves showed less bridging of the cavity of the left ventricle by ramifications of the left branch than the hearts which produced concordant curves.

Further experimental proof is found in the work of Wilson and Herrmann.[29] From time to time series of clinical curves interpreted as examples of bundle-branch block have been published.[1, 2, 8, 9, 13, 18, 27]

It is interesting to note that Cohn and Lewis reported four cases in which the electrocardiographic diagnosis of bundle-branch block was not verified by pathological study. The discrepancy was explained by the assumption that functional changes, as revealed by the electrocardiogram, do not necessarily depend on pathological changes. Oppenheimer and Pardee reported two cases in which lesions were found in the branch which were not indicated by the electrocardiogram.

GRAPHIC CRITERIA OF COMPLETE BUNDLE-BRANCH BLOCK

1. The increased duration of the QRS complex is probably the most reliable indication of complete bundle-branch block. Wilson and Herrmann[29] conclude that the QRS interval is usually more than 0.1 of a second, and often more than 0.15 of a second. They emphasize the point made by Lewis, as a warning against misinterpretation, that the QRS interval of left ventricular preponderance curves is at times more than 0.1 of a second, due to the increased time consumed by the excitation wave in penetrating the hypertrophied walls of the left ventricle. The prolonged QRS interval of complete bundle-branch block is believed to be chiefly due to the retarded spread of the excitation wave through the interventricular septum, and the interval therefore depends on the

thickness of the septum, which is probably considerable in greatly hypertrophied hearts.

2. There is invariably a high amplitude of the QRS complexes in complete bundle-branch block which, according to Wilson and Herrmann[29] is chiefly owing to lack of balance. They call attention to the fact that in normal hearts the effects produced by the activation of the free wall of the left ventricle and those produced by the activation of the free wall of the right ventricle, which are directed oppositely, occur synchronously and tend to neutralize each other. In bundle-branch block these regions are not activated simultaneously, and accordingly a disturbance of the normal balance between them and relatively large complexes result.

3. The T-wave is exaggerated and points in a direction opposite to that of the main deflection of the QRS group.

4. Notching of the QRS complexes often occurs.

5. In the clinical curves of the complete right bundle-branch block the QRS complex is directed upward in Lead I while the T-wave is directed downward, and the reverse occurs in Lead III.

In the clinical curves of complete left bundle-branch block the QRS complex is directed downward in Lead I while the T-wave is directed upward, and the reverse occurs in Lead III.

ABNORMALITIES OF THE QRS COMPLEX NOT CONFORMING TO THE ACCEPTED CRITERIA OF COMPLETE BUNDLE-BRANCH BLOCK

In 1916 Robinson called attention to clinical cases in which the QRS complexes were abnormal and were attributed to probably defective intraventricular conduction.

Oppenheimer and Rothschild reported a group of cases in which changes in the QRS complexes occurred and in which pathological findings indicated involvement of the arborizations of the His bundle. They formulated the view that the changes in the QRS complexes were due to defective conduction through the finer branches of the ventricular conduction system, and introduced the term "arborization block" to describe this disturbance.

In 1919 I published a paper, "Arborization block," in which 138 cases were reported. At this time I accepted the views of Oppenheimer and Rothschild, but in many instances more recent experimental studies throw considerable doubt on the security of these interpretations.

The experimental work of Smith is interesting in this connection. In his early work he found that ligation of various branches of the coronary arteries resulted in extensive lesions of the subendocardial myocardium, but no abnormalities in the QRS complexes of the electrocardiogram. However, in later work, he was able to produce QRS changes after subjecting the heart to unusual strain and incising the endocardium of the left ventricle. He concluded that at least two factors were necessary for

the development of abnormal curves: lesions of the conduction system, and cardiac fatigue.

Wilson and Herrmann[30] satisfactorily explain many of the QRS abnormalities which do not conform to those of complete bundle-branch block. Their work cannot be reviewed here in detail, but they concluded that delayed conduction of the cardiac impulse through one of the main branches of the His bundle gives rise to ventricular complexes that are transitional in form between the normal complex and the complex of complete bundle-branch block. These transitional complexes are presumable evidence of incomplete bundle-branch block.

CLINICAL CASES

This study comprises 105 cases in which electrocardiograms were obtained conforming to those of experimental complete bundle-branch block. From the data available after a critical study of this group it is not possible to prove the actual existence of lesions involving the bundle branches, as only two deaths occurred during my observation. However, the electrocardiograms of these cases so accurately resemble those obtained from experimental lesions that the presence of bundle-branch lesions in most cases is probable.

In ninety-nine cases (94 per cent) the electrocardiograms were of the complete right bundle-branch block type, while in six cases (6 per cent) the left branch type occurred.

The infrequency of complete left bundle-branch block has been ascribed to anatomical differences of the two branches. The right branch passes directly to the large papillary muscle of the right ventricle as an undivided structure, while the left branch divides almost immediately, spreading out in a fan-like manner, rendering complete obstruction unlikely except by a very extensive lesion.

ELECTROCARDIOGRAPHIC FINDINGS

The graphic criteria for complete bundle-branch block now generally accepted (and tabulated earlier in this paper) were rigidly adhered to in the cases under discussion. The QRS interval in all cases was prolonged beyond 0.12 of a second; it was 0.15 of a second in many cases, and even greater in others. It is not possible to reproduce the electrocardiograms of all the cases but the characteristics of these curves are available in the accompanying tables (Tables I to V).

An analysis of the electrocardiograms of the complete right bundle-branch type showed variations in notchings which permitted them to be divided into four groups.

Group I comprises those electrocardiograms displaying little or no notching but in which distinct slurring of the QRS complex occurs. Forty-three curves conformed to this type (Figs. 1 and 7).

TABLE I

GROUP I. COMPLEXES SHOWING RELATIVELY LITTLE NOTCHING, FORTY-THREE CASES (41 PER CENT), COMPLETE RIGHT BUNDLE-BRANCH BLOCK*

CASE	LEAD I AMPLITUDE			LEAD II AMPLITUDE			LEAD III AMPLITUDE			DURATION OF QRS			REMARKS	DIRECTION OF T-WAVE		
	Q	R	S	Q	R	S	Q	R	S	LEAD I	LEAD II	LEAD III		LEAD I	LEAD II	LEAD III
1	0.0	+21.0	−5.5	0.0	+14.5	− 5.5	0.0	+ 2.0	−12.5	0.19	0.19	0.17	Apex notched in Lead III	−	±	+
2	0.0	+18.0	−2.5	0.0	+ 7.5	− 6.5	0.0	+ 3.0	−16.5	0.18	0.16	0.15	Descending limb notched in Lead III	−	+	+
3	0.0	+12.75	0.0	0.0	+ 3.25	−16.25	0.0	+ 2.5	−23.5	0.15	0.16	0.16	Descending limb notched in Leads II and III	−	+	+
4	0.0	+22.75	0.0	0.0	+ 6.0	− 7.5	0.0	?**	?	0.16	0.15	0.15	No notching	−	+	+
5	0.0	+22.0	0.0	0.0	+ 2.0	−14.0	0.0	?	?	0.16	0.12	0.15	No notching	−	+	+
6	0.0	+25.0	0.0	0.0	+12.75	0.0	0.0	0.0	−30.0	0.16	0.11	0.13	No notching	−	+	+
7	0.0	+18.0	−4.5	0.0	+ 9.0	−30.0	0.0	+11.0	?	0.12	0.12	0.12	No notching	−	+	+
8	−1.0	+21.75	−7.75	−1.0	+10.0	−26.0	0.0	+ 4.0	−27.75	0.16	0.13	0.17	No notching	−	−	±
9	0.0	+19.75	0.0	0.0	+13.75	− 6.0	0.0	+ 8.0	?	0.18	0.17	0.15	Descending limb notched in Leads II and III	−	±	+
10	0.0	+16.0	−3.0	0.0	+ 3.0	− 8.25	0.0	?	?	0.15	0.12	0.16	No notching	−	+	+
11	0.0	+21.0	0.0	0.0	+ 9.25	−14.0	0.0	+ 8.0	?	0.16	0.17	0.15	Descending limb notched in Lead II	−	+	+
12	−3.0	+21.0	0.0	0.0	+ 9.0	−10.0	0.0	+10.0	?	0.13	0.14	0.16	No notching	−	+	+
13	0.0	?	0.0	0.0	+10.25	− 7.0	0.0	+ 7.0	−22.0	0.13	0.15	0.14	Apex notched in Lead II	−	+	+
14	0.0	+18.0	0.0	0.0	+ 7.75	−25.0	0.0	+ 3.0	?	0.16	0.17	0.17	No notching	−	+	+
15	0.0	+14.75	0.0	0.0	?	0.0	0.0	+ 3.0	−12.0	0.14	0.16	0.15	Apex notched in Lead III	−	+	+
16	0.0	+15.5	0.0	0.0	+ 5.0	− 5.0	0.0	+ 3.0	−17.75	0.18	0.16	0.17	Apex notched in Lead II	−	+	+
17	0.0	+21.0	0.0	0.0	+20.0	− 3.75	0.0	+ 3.5	?	0.13	0.13	0.13	No notching	−	+	+
18	0.0	+22.75	−8.0	0.0	+15.0	−11.0	0.0	+ 4.0	?	0.16	0.15	0.12	No notching	−	+	+
19	0.0	+37.0	−6.0	0.0	+23.0	−13.75	0.0	+ 3.75	?	0.19	0.17	0.19	No notching	−	+	+
20	0.0	+16.0	0.0	0.0	+ 5.75	− 9.0	0.0	+ 4.75	?	0.14	0.14	0.14	No notching	−	+	+
21	0.0	+19.75	0.0	0.0	+16.0	− 1.75	0.0	+ 6.0	−10.0	0.19	0.16	0.16	No notching	−	−	+
22	0.0	+19.0	0.0	0.0	+ 5.75	−24.5	0.0	+ 4.75	?	0.14	0.15	0.15	Apex notched in Lead II	−	+	+

TABLE I—CONT'D

CASE	LEAD I AMPLITUDE			LEAD II AMPLITUDE			LEAD III AMPLITUDE			DURATION OF QRS			REMARKS	DIRECTION OF T-WAVE		
	Q	R	S	Q	R	S	Q	R	S	LEAD I	LEAD II	LEAD III		LEAD I	LEAD II	LEAD III
23	0.0	+22.5	0.0	0.0	+ 5.25	−12.0	0.0	+ 3.0	?	0.16	0.15	0.14	No notching	−	+	+
24	0.0	+15.5	0.0	0.0	+ 2.0	−20.75	0.0	?	?	0.16	0.16	0.17	Apex notched in Lead II	−	+	+
25	0.0	+16.0	0.0	0.0	+ 5.75	− 4.5	0.0	+ 2.5	−17.5	0.20	0.19	0.17	Apex notched in Lead III	−	+	+
26	0.0	+18.0	0.0	0.0	+12.25	− 4.0	−1.5	+ 4.0	−12.0	0.16	0.14	0.16	Apex notched in Lead II	−	+	+
27	0.0	+20.5	0.0	0.0	+12.5	− 5.25	0.0	+ 2.5	−16.5	0.15	0.16	0.14	Apex notched in Lead III	−	+	+
28	0.0	+20.0	0.0	0.0	+ 4.75	−14.5	0.0	+ 3.0	?	0.16	0.14	0.16	Descending limb notched in Lead II	−	+	+
29	0.0	+20.0	0.0	0.0	+14.0	−10.0	0.0	+ 2.5	?	0.16	0.14	0.15	No notching	−	+	+
30	0.0	+15.0	0.0	0.0	+ 4.75	− 3.25	0.0	+ 2.0	?	0.15	0.12	0.14	Apex notched in Lead II	−	+	+
31	0.0	+27.25	0.0	0.0	+ 5.25	−15.0	0.0	+ 4.0	?	0.15	0.15	0.16	No notching	−	+	+
32	0.0	+14.25	0.0	0.0	+ 6.0	−13.25	0.0	+ 3.0	?	0.12	0.13	0.14	No notching	−	+	+
33	0.0	+13.25	0.0	0.0	+ 8.0	− 8.25	0.0	+ 5.5	−15.0	0.16	0.16	0.14	Apex notched in Lead II	−	+	+
34	0.0	+16.0	0.0	0.0	+ 4.5	−12.50	0.0	+ 4.75	?	0.13	0.14	0.14	Apex notched in Lead II	−	+	+
35	0.0	+15.5	0.0	0.0	+ 6.5	− 3.0	0.0	+ 6.5	−19.5	0.16	0.17	0.16	Apex notched in Lead II	−	−	+
36	0.0	+18.0	0.0	0.0	+ 9.0	0.0	0.0	+ 2.5	−12.5	0.16	0.16	0.12	Ascending and descending limbs notched in Lead II	−	±	+
37	0.0	+11.25	0.0	0.0	+ 6.5	0.0	0.0	+ 5.0	− 8.5	0.16	0.16	0.16	Apex notched in Leads II and III	−	+	+
38	0.0	+20.0	0.0	0.0	+ 6.5	− 2.0	0.0	+ 4.25	?	0.16	0.15	0.15	Apex notched in Lead II	−	+	+
39	0.0	+15.0	0.0	0.0	+ 8.0	−12.5	0.0	+ 7.5	−20.0	0.12	0.15	0.15	Descending limb notched in Lead II	−	+	+
40	0.0	?	0.0	0.0	+ 8.25	−18.0	0.0	+ 5.0	?	0.16	0.16	0.15	Descending limb notched in Lead II	−	+	+
41	0.0	?	0.0	0.0	+21.0	0.0	0.0	+ 3.0	?	0.16	0.15	0.16	No notching	−	+	+
42	0.0	+26.0	0.0	0.0	+12.5	0.0	0.0	+ 2.0	−14.75	0.14	0.14	0.14	Descending limb notched in Lead III	−	−	+
43	0.0	+18.75	0.0	0.0	+ 4.75	− 6.75	0.0	+ 2.75	−20.0	0.18	0.17	0.18	Apex notched in Lead II	−	+	+

**In all cases in which the amplitude of complexes are marked (?) the deflections were so great that they were recorded in part only.
*Measurements are all averages of several readings.

TABLE II

GROUP II. COMPLETE RIGHT BUNDLE-BRANCH BLOCK, TWENTY-TWO CASES (21 PER CENT). COMPLEXES WITH NOTCHING OF THE APEX

CASE	LEAD I AMPLITUDE			LEAD II AMPLITUDE			LEAD III AMPLITUDE			DURATION OF QRS			REMARKS	DIRECTION OF T-WAVE		
	Q	R	S	Q	R	S	Q	R	S	LEAD I	LEAD II	LEAD III		LEAD I	LEAD II	LEAD III
1	0.0	+17.75	0.0	0.0	+ 6.0	− 8.0	0.0	+ 2.0	−21.0	0.14	0.15	0.15	Apex in Lead I and descending limbs in Leads II and III notched	−	+	+
2	0.0	+31.0	0.0	0.0	+ 3.0	−14.5	0.0	+ 2.25	?	0.16	0.15	0.16	Apex in Lead I and descending limb in Lead II notched	−	+	+
3	0.0	+21.0	0.0	0.0	+17.0	− 6.25	0.0	+ 8.0	−13.25	0.15	0.15	0.14	Apex in Leads I and II and descending limb in Lead III notched	−	+	+
4	0.0	+17.0	0.0	0.0	+ 5.0	− 5.25	0.0	+ 4.0	?	0.14	0.14	0.13	Apex in Lead I and descending limb in Lead II notched	−	+	+
5	0.0	+16.25	0.0	0.0	+ 6.5	−13.0	0.0	+ 2.25	?	0.16	0.16	0.15	Apex in Lead I notched	−	±	+
6	0.0	+16.0	0.0	0.0	+ 9.75	0.0	0.0	+ 4.0	−10.75	0.15	0.15	0.14	Apex in all leads notched	−	+	+
7	0.0	+12.75	0.0	0.0	+12.25	− 2.25	0.0	+ 5.0	−11.0	0.14	0.13	0.13	Apex in Lead I and ascending limb in Lead II notched	−	+	+
8	0.0	+17.0	0.0	0.0	+11.0	−14.0	0.0	+ 6.0	?	0.14	0.14	0.15	Apex in Lead I notched	−	+	+
9	0.0	+13.25	0.0	0.0	+ 7.0	0.0	0.0	+ 2.75	−12.0	0.15	0.14	0.14	Apex in all leads notched	−	+	+
10	0.0	+18.0	0.0	0.0	+19.25	0.0	−4.0	+12.0	− 9.25	0.18	0.18	0.16	Apex in Leads I and II notched	−	±	+
11	0.0	+16.0	0.0	0.0	+ 7.0	− 5.75	0.0	+ 2.0	−15.75	0.13	0.14	0.14	Apex in all leads notched	−	+	+
12	0.0	+22.0	0.0	0.0	+ 8.0	− 4.75	0.0	+ 3.0	?	0.14	0.14	0.14	Apex in Leads I and II notched	−	+	+
13	0.0	+18.0	0.0	0.0	+ 3.75	−10.75	0.0	+ 2.25	−16.0	0.18	0.18	0.18	Apex in Lead I and descending limb in Lead III notched	−	+	+
14	0.0	+14.0	0.0	0.0	+11.75	0.0	0.0	+ 4.0	− 8.75	0.17	0.14	0.15	Apex in Leads I and II notched	−	+	+
15	0.0	+18.0	0.0	0.0	+10.0	− 6.5	0.0	+ 4.0	−16.25	0.17	0.15	0.16	Apex in Lead I notched	−	+	+
16	0.0	+13.0	0.0	0.0	+ 7.0	− 3.0	0.0	+ 3.0	?	0.15	0.14	0.14	Apex in Leads I and II and descending limb in Lead III notched	−	±	+
17	0.0	+ 9.0	0.0	0.0	+11.5	− 9.0	0.0	+ 9.0	−13.0	0.14	0.15	0.15	Apex notched in all leads	−	+	+
18	0.0	+ 9.75	0.0	0.0	+ 4.75	− 6.25	0.0	− 3.25	−10.5	0.13	0.14	0.13	Apex in Leads I and III and descending limb in Lead II notched	−	+	+
19	0.0	+ 5.5	0.0	0.0	+ 7.0	− 5.0	0.0	+ 4.5	− 9.5	0.17	0.16	0.16	Apex notched in all leads	−	+	+
20	0.0	+10.5	0.0	0.0	+13.0	0.0	0.0	+ 8.0	− 9.75	0.15	0.14	0.15	Apex in Lead I notched	−	+	+
21	0.0	+ 4.5	−3.0	0.0	+ 5.0	−15.0	0.0	+ 2.75	−15.5	0.14	0.16	0.16	Apex in Leads I and II notched	−	+	+
22	0.0	+13.25	0.0	0.0	+ 7.0	−10.25	0.0	+ 5.0	−16.0	0.15	0.15	0.15	Apex in Lead I and descending limb in Lead III notched	−	+	+

TABLE III

GROUP III. COMPLEXES SHOWING NOTCHING OF THE DESCENDING LIMB, THIRTY-ONE CASES (30 PER CENT). COMPLETE RIGHT BUNDLE-BRANCH BLOCK

CASE	LEAD I AMPLITUDE			LEAD II AMPLITUDE			LEAD III AMPLITUDE			DURATION OF QRS			REMARKS	DIRECTION OF T-WAVE		
	Q	R	S	Q	R	S	Q	R	S	LEAD I	LEAD II	LEAD III		LEAD I	LEAD II	LEAD III
1	0.0	+24.0	0.0	0.0	+13.75	− 4.75	0.0	+2.5	−19.0	0.17	0.16	0.17	Descending limbs in Leads I and III and apex in Lead II notched	−	+	+
2	0.0	+17.0	0.0	0.0	+ 5.75	− 6.75	0.0	+3.0	−17.5	0.16	0.17	0.16	Descending limb notched in all leads	−	+	+
3	0.0	+13.0	0.0	0.0	+ 8.0	− 8.0	0.0	+3.75	−11.0	0.15	0.16	0.16	Descending limbs in Leads I and III and apex in Lead II notched	−	±	+
4	0.0	+17.25	0.0	0.0	+ 7.75	0.0	0.0	+1.75	−13.0	0.16	0.14	0.14	Descending limbs in Leads I and II and apex in Lead III notched	−	−	+
5	0.0	+16.5	0.0	0.0	+ 7.0	?	0.0	+5.0	?	0.16	0.16	0.16	Descending limbs notched in all leads	−	+	+
6	0.0	+10.25	0.0	0.0	+ 4.25	− 5.5	0.0	+4.0	− 7.0	0.17	0.17	0.17	Descending limbs in Leads I and II and apex in Lead III notched	−	+	+
7	0.0	+12.25	0.0	0.0	+ 5.0	− 6.0	0.0	+5.0	−12.5	0.16	0.15	0.16	Descending limb notched in Lead I	−	+	+
8	0.0	+10.75	0.0	0.0	+ 6.0	−19.0	0.0	+5.75	−25.0	0.13	0.14	0.15	Descending limbs notched in all leads	−	+	+
9	0.0	+12.25	0.0	0.0	+ 4.0	−14.0	0.0	+3.75	?	0.16	0.14	0.15	Descending limbs in Leads I and III and apex in Lead II notched	−	+	+
10	0.0	+18.5	0.0	0.0	+ 8.0	− 9.0	0.0	+3.5	−19.0	0.17	0.16	0.17	Descending limb notched in Lead I	−	+	+
11	0.0	+23.0	0.0	0.0	+ 5.0	−14.5	0.0	+4.0	?	0.15	0.14	0.15	Descending limbs notched in Leads I and II	−	+	+
12	0.0	+19.0	0.0	0.0	+13.0	− 6.5	0.0	+4.0	−16.5	0.14	0.14	0.14	Descending limbs notched in Leads I and III	−	−	+
13	0.0	+12.5	0.0	0.0	+11.75	− 4.0	0.0	+6.0	− 8.75	0.14	0.13	0.14	Descending limb in Lead I and apex in Lead III notched	−	+	+
14	0.0	+18.0	−4.75	0.0	+ 9.25	− 3.75	0.0	+3.5	?	0.14	0.15	0.15	Descending limbs in Lead I and apex in Lead II notched	−	+	+
15	0.0	+27.0	0.0	0.0	+ 5.75	−19.0	0.0	+3.75	?	0.20	0.19	0.20	Descending limb in Lead I and ascending limb in Lead III notched	−	+	+

TABLE III—CONT'D

CASE	LEAD I AMPLITUDE			LEAD II AMPLITUDE			LEAD III AMPLITUDE			DURATION OF QRS			REMARKS	DIRECTION OF T-WAVE		
	Q	R	S	Q	R	S	Q	R	S	LEAD I	LEAD II	LEAD III		LEAD I	LEAD II	LEAD III
16	0.0	+14.5	0.0	0.0	+11.75	− 3.0	0.0	+3.0	− 9.0	0.15	0.15	0.15	Descending limbs in Leads I and III notched	−	−	+
17	0.0	+13.0	0.0	0.0	+ 8.0	−15.0	0.0	+2.75	−17.0	0.17	0.16	0.16	Descending limbs in all leads notched	−	+	+
18	0.0	+13.5	0.0	0.0	+ 8.0	− 3.0	0.0	+3.5	−11.0	0.12	0.13	0.13	Descending limbs in Leads I and III notched	−	+	+
19	0.0	+13.0	0.0	0.0	+ 6.75	− 8.0	0.0	+5.75	−19.0	0.17	0.17	0.16	Descending limbs in Leads I and II and apex in Lead III notched	−	+	+
20	0.0	+20.0	0.0	0.0	+ 9.0	− 9.75	0.0	+2.75	−19.5	0.17	0.18	0.17	Descending limbs in Leads I and III and apex in Lead II notched	−	+	+
21	0.0	+16.75	0.0	0.0	+14.0	−11.25	0.0	+3.5	−17.0	0.17	0.16	0.17	Descending limbs in all leads notched	−	−	+
22	0.0	+13.75	0.0	0.0	+10.25	− 2.25	0.0	+6.0	−11.0	0.15	0.13	0.15	Descending limbs in Leads I and III and apex in Lead II notched	−	±	+
23	0.0	+17.5	0.0	0.0	+16.0	− 7.0	0.0	+7.0	−12.0	0.16	0.14	0.15	Descending limbs in Leads I and III notched	−	−	+
24	0.0	+19.0	0.0	0.0	+ 7.5	− 7.75	0.0	+5.25	−15.0	0.16	0.16	0.16	Descending limbs in Leads I and II and apex in Lead III notched	−	+	+
25	0.0	+17.5	0.0	0.0	+17.0	− 4.75	0.0	+5.25	?	0.15	0.14	0.14	Descending limbs in Leads I and III notched	−	+	+
26	0.0	+15.0	0.0	0.0	+ 7.0	− 6.0	0.0	+5.25	−11.5	0.16	0.14	0.16	Descending limbs in Leads I and II apex in Lead III notched	−	+	+
27	0.0	+13.0	0.0	0.0	+ 6.0	−10.0	0.0	+4.0	−16.5	0.14	0.17	0.16	Descending limbs in all leads notched	−	+	+
28	0.0	+15.0	0.0	0.0	+ 8.0	− 4.0	0.0	+2.0	−13.0	0.14	0.16	0.15	Descending limb in Lead I and apex in Lead III notched	−	+	+
29	0.0	+22.5	0.0	0.0	+17.0	− 7.0	0.0	+2.5	?	0.15	0.16	0.15	Descending limbs notched in Leads I and II	−	+	+
30	0.0	+13.0	0.0	0.0	+ 6.5	− 6.25	0.0	+4.5	−11.5	0.14	0.14	0.14	Descending limbs in all leads notched	−	±	+
31	0.0	+15.0	0.0	0.0	+ 8.0	−14.25	0.0	+4.25	?	0.17	0.16	0.15	Descending limb in Lead I and descending and ascending limbs in Lead II notched	−	+	+

TABLE IV

GROUP IV. COMPLEXES SHOWING NOTCHING OF THE ASCENDING LIMB, THREE CASES (3 PER CENT). COMPLETE RIGHT BUNDLE-BRANCH BLOCK

CASE	LEAD I AMPLITUDE			LEAD II AMPLITUDE			LEAD III AMPLITUDE			DURATION OF QRS			REMARKS	DIRECTION OF T-WAVE		
										LEAD	LEAD	LEAD		LEAD	LEAD	LEAD
	Q	R	S	Q	R	S	Q	R	S	I	II	III		I	II	III
1	0.0	+14.5	0.0	0.0	+5.75	−18.5	0.0	+3.5	?	0.15	0.15	0.15	Ascending limb in all leads notched	−	+	+
2	0.0	+15.0	0.0	0.0	+8.0	− 3.0	0.0	+4.5	−14.0	0.16	0.14	0.15	Ascending limb in Lead I and descending limbs in Leads II and III notched	−	+	+
3	0.0	+18.0	0.0	0.0	+5.75	− 2.0	0.0	+2.0	−18.0	0.16	0.15	0.16	Ascending limbs in Leads I and II and descending limb in Lead III notched	−	+	+

TABLE V

COMPLETE LEFT BUNDLE-BRANCH BLOCK, SIX CASES (6 PER CENT)

CASE	LEAD I AMPLITUDE			LEAD II AMPLITUDE			LEAD III AMPLITUDE			DURATION OF QRS			REMARKS	DIRECTION OF T-WAVE		
										LEAD	LEAD	LEAD		LEAD	LEAD	LEAD
	Q	R	S	Q	R	S	Q	R	S	I	II	III		I	II	III
1	0.0	+10.0	−18.0	−5.0	+24.0	0.0	−7.0	+32.5	0.0	0.16	0.15	0.16	Ascending limb in Lead I and descending limbs in Leads II and III notched	+	−	−
2	0.0	+ 6.5	−19.5	0.0	+27.75	−2.0	−3.0	+28.0	0.0	0.17	0.15	0.15	Descending limb in Lead II notched	+	−	−
3	0.0	+ 7.0	**− 5.5**	0.0	+14.75	−6.75	−3.5	+24.75	−5.5	0.15	0.16	0.14	Apex in Leads I and II and descending limb in Lead III notched	+	−	−
4	0.0	+12.5	− 8.5	0.0	+20.5	**−5.25**	−3.25	+26.5	0.0	0.15	0.14	0.14	Ascending limb in Lead I and descending limbs in Leads II and III notched	±	−	−
5	0.0	+ 4.5	−17.25	−8.0	+16.5	0.0	−7.0	+33.0	0.0	0.16	0.16	0.15	Apex notched in all leads	+	±	−
6	0.0	+ 4.0	− 2.0	0.0	+11.75		0.0	+15.0	0.0	0.14	0.13	0.14	Ascending limb notched in all leads	+	−	−

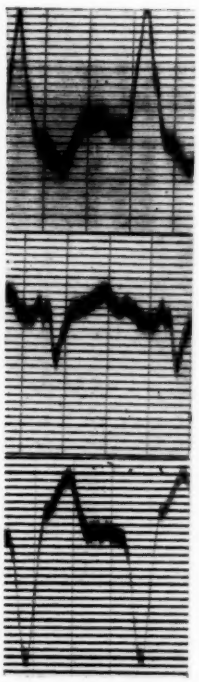

Fig. 1.

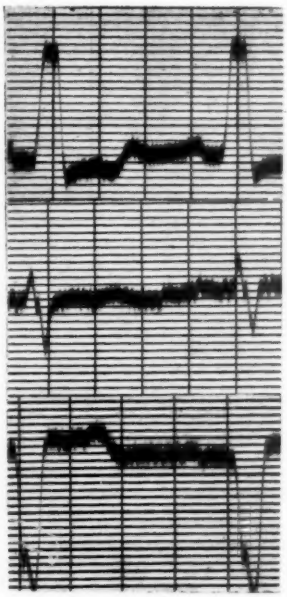

Fig. 2.

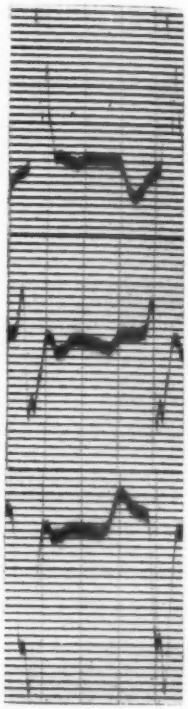

Fig. 3.

Fig. 1.—Complete right bundle-branch block. Group I type.
Fig. 2.—Complete right bundle-branch block. Group II type.
Fig. 3.—Complete right bundle-branch block. Group III type.

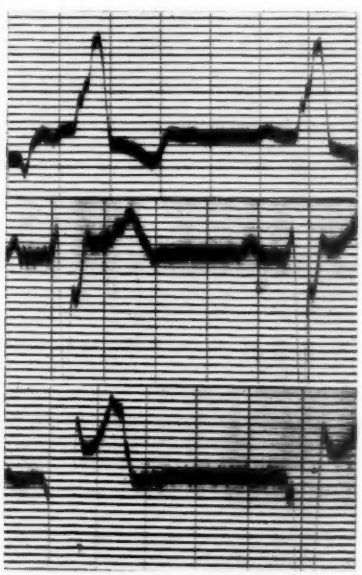

Fig. 4. Complete right bundle-branch block. Group IV type.

Group II records twenty-two curves in which notching dominantly involves the apex of the QRS complex (Figs. 2 and 8).

Group III is composed of thirty-one curves revealing notching chiefly affecting the descending limb of the complex (Figs. 3 and 9).

Group IV is small, comprising only three cases in which the notching was mainly confined to the ascending limb of the complex (Figs. 4 and 10).

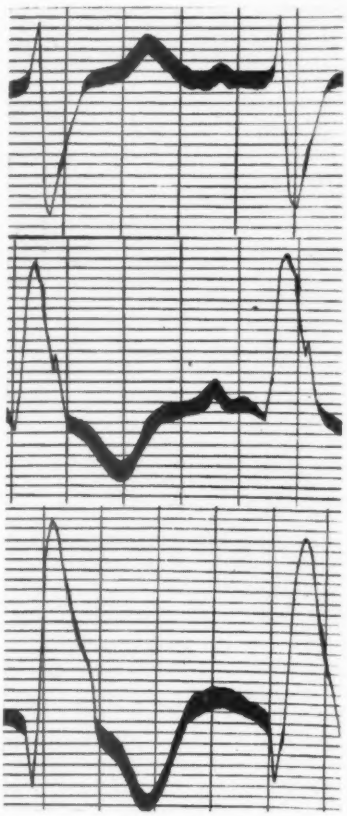

Fig. 5.

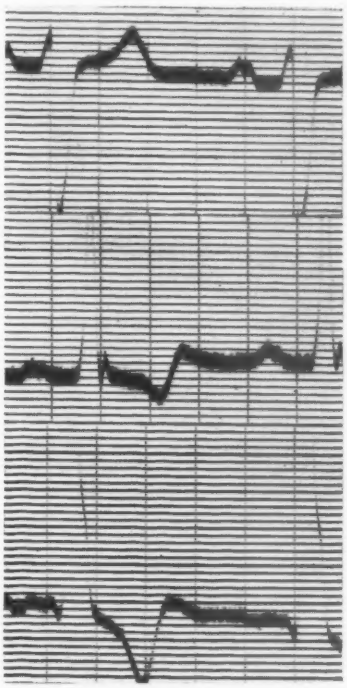

Fig. 6.

Fig. 5.—Complete left bundle-branch block.
Fig. 6.—Complete left bundle-branch block.

Examples of complete left bundle-branch block are shown in Figs. 5, 6 and 11.

Only four instances (4 per cent) of auricular fibrillation occurred in the whole group, being equally distributed among the right and the left branch curves. There were three tracings (3 per cent) showing prolongation of the P-R interval beyond the normal. This incidence is even smaller than is usually encountered in a large group of abnormal electrocardiograms.

CLINICAL FEATURES

There were thirty-eight cases in the sixth decade and forty in the seventh. Nine patients were more than seventy years of age, and three less than thirty; the youngest was seventeen and the oldest eighty-eight. Sixty of the patients were males, and forty-five were females.

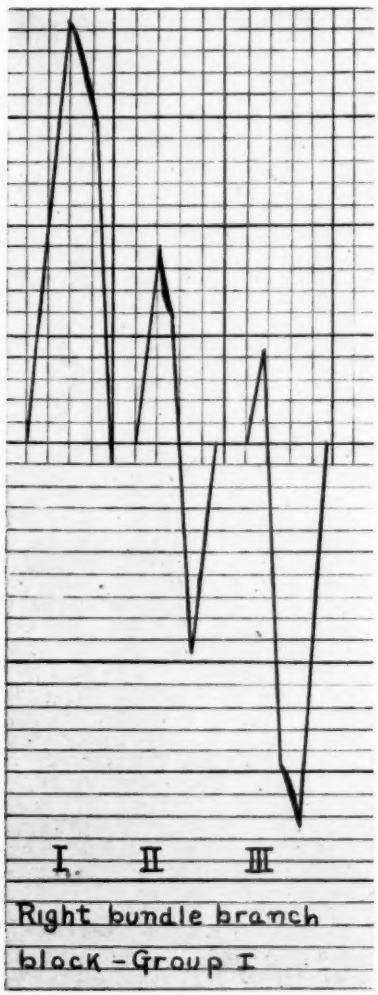

Fig. 7.

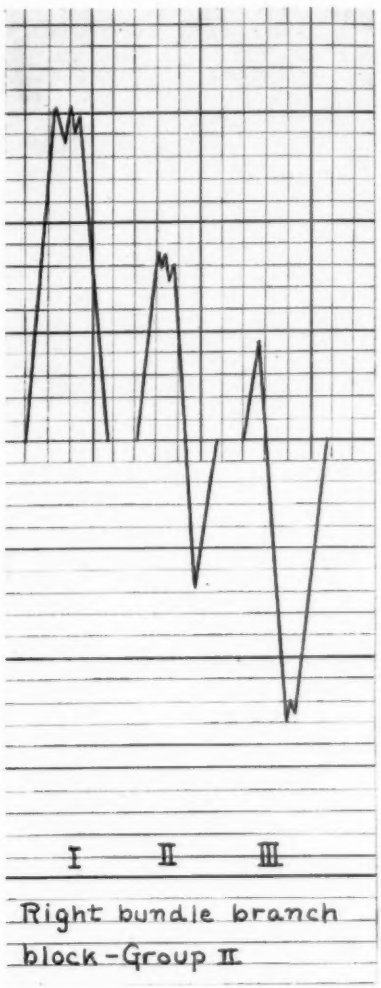

Fig. 8.

Fig. 7.—Composite curve of forty-three electrocardiograms representing Group I.
Fig. 8.—Composite curve of twenty-two electrocardiograms representing Group II.

Probable Types of Cardiac Disease.—Myocardial disease associated with hypertension occurred with greatest frequency, being recorded in fifty-five cases (52 per cent). It is difficult to estimate the degree of associated arteriosclerotic change in the hypertensive heart, and it therefore seemed best to group these cases as above. The incidence of hyper-

tensive heart disease was greater than in the series of Herrick and Smith, and of White and Viko. Arteriosclerotic myocardial disease occurred in forty-three cases (41 per cent).

There were only four cases (4 per cent) of cardiovascular syphilis, and in all instances the lesion was that of aortitis associated with aortic insuf-

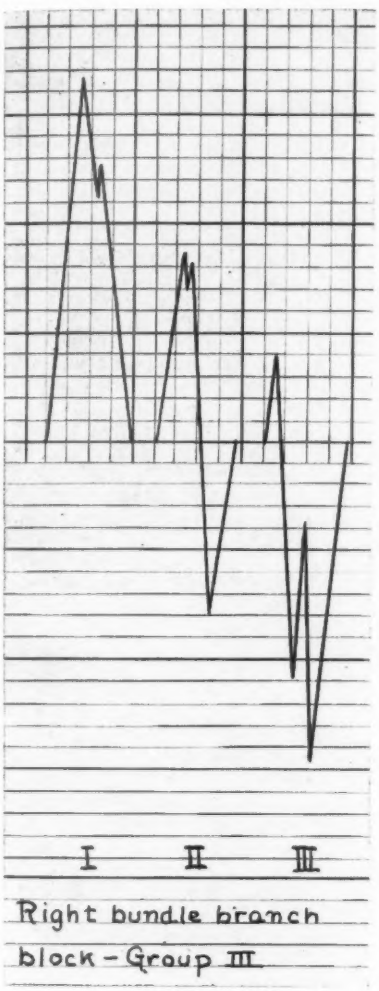

Fig. 9.

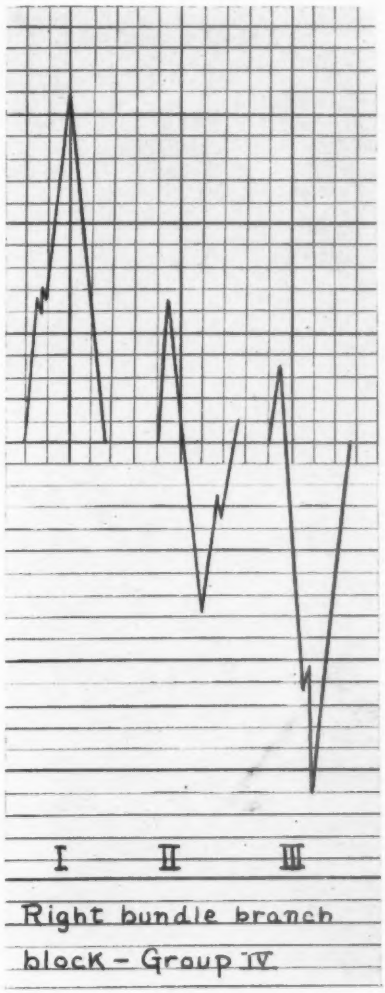

Fig. 10.

Fig. 9.—Composite curve of thirty-one electrocardiograms representing Group III.
Fig. 10.—Composite curve of three electrocardiograms representing Group IV.

ficiency. The blood Wassermann reactions in these cases were strongly positive. The incidence of syphilis was definitely less than in previously reported cases in which it was 7 per cent,[26] 14 per cent,[7] and 20 per cent.[6]

The incidence of rheumatic heart disease was very small, only two cases (2 per cent) being recorded. In one case there occurred a chronic

rheumatic pancarditis with involvement of the mitral and aortic valves, and in another case aortic insufficiency was present. Cases in which there was a history of rheumatic infection occurred in from 10 per cent[26] to 29 per cent[7] in other published studies. Congenital heart disease occurred in one case (1 per cent).

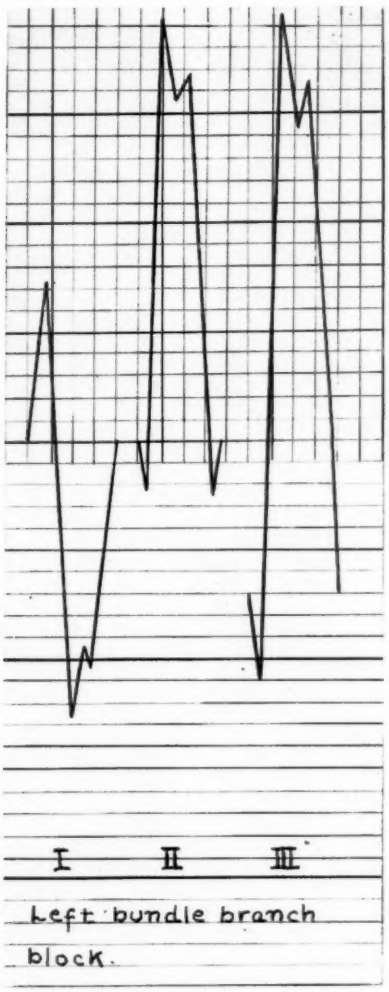

Fig. 11.—Composite curve of six electrocardiograms.

Symptoms.—Dyspnea on exertion was complained of in all cases, and orthopnea occurred in twelve cases (11 per cent). The syndrome of congestive heart failure occurred in fourteen cases (13 per cent). It is interesting to note that most of the patients were ambulatory without great difficulty. The anginal syndrome was present in thirty-one cases (30 per cent) and in one the history was very suggestive of a previous myocardial infarction.

Cardiac Findings.—The area of cardiac dullness was increased in all cases. In thirty-three cases the heart tones were distant; they lacked definition and differentiation and were often described as being tick-

TABLE VI

DURATION OF CARDIAC SYMPTOMS PRIOR TO EXAMINATION

DURATION, MONTHS	CASES	DURATION, YEARS	CASES
3	1	4	4
4	2	5	5
6	9	6	4
8	1	7	3
10	1	9	1
12	15	10	2
14	1	11	1
18	5	15	1
24	22	20	1
36	12	28*	1

*Case of congenital heart disease.

TABLE VII

MORTALITY RATE

YEARS SINCE EXAMINATION	TOTAL CASES	PATIENTS TRACED	CARDIAC DEATHS			DEATHS FROM OTHER CAUSES	PATIENTS LIVING	
			NO.	%	AVERAGE TIME SINCE EXAMINATION, MONTHS		NO.	%
10	4	3	2	67	14	1	0	0
9	7	6	5	83	19	1	0	0
8	8	8	8	100	14	0	0	0
7	9	9	8	89	13	1	0	0
6	14	9	7	78	18	0	2	22
5	10	5	3	60	32	1	1	20
4	5	3	3	100	9	0	0	0
3	5	3	0	0		0	3	100
2	12	5	4	80	5	1	0	0
1	31	15	3	20	5	0	12	80
Total group averages	105	66	43	65	14	5	18	27

TABLE VIII

COMPOSITE MORTALITY RATE (PUBLISHED CASES)

AUTHORS	TOTAL CASES	PATIENTS TRACED	CARDIAC DEATHS			DEATHS FROM OTHER CAUSES	PATIENTS LIVING	
			NO.	%	AVERAGE TIME SINCE EXAMINATION, MONTHS		NO.	%
Herrick and Smith	35	21	10	48	18	1	10	48
White and Viko	41	36	22	61	9	4	10	28
Hart	25	21	18	86	9	0	3	14
Willius	105	66	43	65	14	5	18	27
Total	206	144	93	65	12	10	41	28

tack in character. Gallop rhythm was noted in nine cases. A systolic apical murmur was present in twenty-nine cases, an aortic systolic murmur in eight, and systolic murmurs at both the apex and the aortic area in five.

Preceding History of Cardiac Disease.—Symptoms referable to the heart had generally existed for from one to three years. In twelve cases the duration was six months or less, and in one case symptoms had lasted twenty years (Table VI).

Life Expectancy.—Information has been obtained regarding the condition of sixty-six patients (63 per cent). It was found that in forty-three patients (65 per cent) who died from cardiac disease, the average length of life after examination at the clinic was fourteen months. Five died from causes other than heart disease. Eighteen (27 per cent) are known to be alive. In general the death rate was highest among patients who had been seen four or more years before (Table VII).

No clear evidence exists that the findings of complete bundle-branch block per se influence cardiac mortality except that, as a rule, these graphic abnormalities occur in patients with quite advanced and extensive cardiac disease.

Carter says: "An organic lesion confined to one branch of the A-V bundle is hardly one which can be regarded as jeopardizing life. We find that a bundle-branch lesion may be present for many years, and the heart still show general efficiency."

Two of the patients in this series who are known to have had these graphic changes six years ago are still alive.

The composite data of previous publications and the results obtained in this study are tabulated in Table VIII. In 144 cases traced, the cardiac mortality was 65 per cent at an average of about twelve months after examination.

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A SUPERNORMAL PHASE IN CONDUCTION AND A RECOVERY CURVE FOR THE HUMAN JUNCTIONAL TISSUES*

RICHARD ASHMAN, PH.D., AND GEORGE R. HERRMANN, M.D.
NEW ORLEANS, LA.

RECENT studies have thrown much light upon the interesting subject of conduction in heart muscle. Experimental studies and a clinical report have shown phenomena in cardiac conduction that closely parallel physiological phenomena exhibited in cases of the excitability and conductivity of nervous tissue.

The two cases of heart-block herein reported are of particular interest because of (*a*) the presence of peculiarities in conduction attributable to a supernormal phase in conduction; (*b*) tremendous prolongation of conduction time beyond the normal in our second case; (*c*) the unusually clear relation between conduction time and the duration of the rest interval of the conducting tissues, a circumstance which makes possible the graphic representation of a recovery curve of conduction in the human heart; and (*d*) the occurrence of unusual relations between the duration of the ventricular cycle, the amplitude of the chief ventricular deflection, and the spread of the QRS group.

THE SUPERNORMAL PHASE

Adrian and Lucas[1] reported that, following the transmission of an impulse through a nerve, there was a period when the nerve was more excitable than when at rest. Thus a stimulus too weak to excite the resting nerve would set up a second impulse if applied from 0.01 to 0.03 or 0.04 second after a previous impulse had been transmitted. This period during which there was an overswing of excitability they termed the *supernormal phase*. Lucas demonstrated further that an impulse set up during the supernormal phase might pass a block in the nerve which the normal impulse could not pass. Thus not only was *excitability* higher than the resting excitability, but *conductivity* was also higher. Recent attempts to show that the velocity of the nerve impulse is greater during the supernormal phase have yielded negative results.[2] Adrian[3] later found that the necessary condition for the appearance of the supernormal phase, both in nerve and in the cold-blooded heart, was a hydrogen-ion concentration of the solution bathing the tissue at or on the acid side of neutrality, although he obtained evidence that for the frog's ventricular muscle the supernormal phase appeared not only when the perfusate was on the acid

*From the Department of Physiology and of Medicine, Tulane University.

side of neutrality but also when it was faintly alkaline. A further important point which Adrian demonstrated was that the excitability during the height of the supernormal phase, while high as compared with the *depressed* resting excitability of the same tissue at the same hydrogen-ion concentration, did not, as a matter of fact, quite reach the excitability of the same tissue in a faintly alkaline solution. Thus the *supernormality* is *relative*, not *absolute*.

In the heart muscle of the turtle a supernormal phase in conduction may be induced by injury, e.g., by compression between the auricles and the ventricle or between parts of the excised ventricle.[10] The phenomenon usually appears when, in addition to injury, there is considerable fatigue of the injured muscle. Under these circumstances there is a time interval, shortly following the transmission of a previous impulse through the compressed muscle, when an impulse will be transmitted more rapidly than it would have been earlier or later. The following figures are taken from unpublished electrograms and illustrate the phenomenon. The first number of each pair is the auricular rest period (cycle length) in seconds; the second number, in parenthesis, the A-V conduction time in seconds for the auricular impulse which terminates the rest period: 1.00 (blocked); 1.50 (blocked); 1.90 (1.04); 2.03 (0.96); 2.50 (0.92); 4.00 (0.85); 4.60 (0.84); 5.10 (0.84); 7.40 (0.86); 30.00 (0.88). The following figures are the rest intervals and conduction times, not from auricle to ventricle, but between the two parts of a ventricular strip, separated by a compression block: 1.00 (blocked); 1.60 (0.59); 2.20 (0.53); 4.00 (0.50); 9.00 (0.55); 20.00 (0.572). Thus the phenomenon is shown equally by the auricular and ventricular muscle of the turtle. A further fact which deserves emphasis is that if the degree of compression is great enough, only those impulses arriving at the compressed muscle during the supernormal phase may be transmitted; earlier impulses, as well as those arriving later, that is, after a *longer rest interval*, are blocked.[16] This latter statement is based not upon one or two, but upon repeated observations. It is an experimental result which deserves emphasis because the human cases reported are of this type.

A supernormal phase in conduction has not yet been described for the mammalian heart, other than that of man, but we are convinced that it will be demonstrated experimentally in the mammal when the right conditions are obtained, for it is a phenomenon manifested by the excitability, contractility or conductivity of such diverse tissues as nerve fibers, skeletal muscle, and cardiac muscle, both auricular and ventricular.

In the clinical literature a number of cases have been described which possibly or probably illustrate the supernormal phase. Vin-

nis[4] reported a case of the treppe or staircase phenomenon following the postextrasystolic pause in the human ventricles. The treppe, Adrian ascribes to the supernormal phase manifesting itself as a stepping up in the contraction of the muscle. Cases of heart-block with salient features similar to those of Case I have been reported by Wilkinson,[5] Starling,[6] Lewis,[7] Mackintosh and Falconer,[8] and others, but the interpretation we offer was not applied. Lewis and Master[9] published reports of two clinical cases which they believed were to be explained on the basis of the supernormal phase. The interpretation of the second case was questioned in a paper by Ashman,[10] but certain points of resemblance between it and our second case now lead us to believe that their conclusion is valid. The first case seems to us to demonstrate clearly a supernormal phase. Their electrocardiograms show a typical complete block interrupted at intervals by premature ventricular responses to auricular systoles. Auricular impulses, corresponding to P-waves arising during an interval of from 0.425 to about 0.700 second after the idioventricular R-wave, were transmitted and produced the early ventricular responses. A slight overlapping of blocked and of transmitted impulses occurred at the end of this range; that is, several impulses slightly earlier than 0.700 second after the R were blocked; one slightly later impulse was transmitted. The conduction time for the impulse arising 0.425 second after the R was 0.12 second, which Lewis and Master stated may indicate an absolutely rather than a relatively supernormal phase. The transmitted impulse, arising slightly later than 0.700 second after the R, was transmitted in 0.168 second. Thus, somewhat irregularly, the transmission times became longer as the rest intervals lengthened. This observation strengthens their contention for a supernormal phase.

It must be noted that at the time their paper was published there was little or no experimental evidence, beyond some old observations by Engelmann[11] on the frog heart and a short paper by Skramlik[12] on the turtle heart, which might have indicated that conduction in the heart is influenced by a supernormal phase. Thus Wolferth,[13] in reporting in abstract a case resembling Lewis and Master's first case, suggested that they based their interpretation upon insufficiently established assumptions. The papers by Ashman may remove this objection. Wolferth speaks of the period or condition of conduction when the impulses pass as a *recovery phase*. The phenomenon under discussion is a distinctly *atypical* recovery phase. All excitable tissues, after response to stimulation, pass through a recovery phase or period during which their excitability and conductivity return to a normal or resting level. If, as Lewis and Master express it, there is an *overswing* in this recovery with subsequent depression, either the

term *supernormal phase* first employed by Adrian and Lucas should be used or a new term should be invented. Lewis and Master's description, an *overswing* in excitability or conductivity, might be used.

CASE I.—*Adams-Stokes Attacks Due to a Sudden Change from Fairly Normal Conduction Time to Complete Heart-Block.* This case has been previously reported as Case 4 in a series of "Some Unusual Disturbances of the Mechanism of the Heart Beat," by Professor Frank N. Wilson and one of us,[14] and through the courtesy and cooperation of Dr. Wilson and his staff the films have been entrusted to us for this more detailed study.

The following history notes were abstracted from the previous report:

T. H., an American coal miner, aged thirty-nine years, came to the medical outpatient department of the University of Michigan Hospital April 4, 1922, complaining of pain in the right upper abdomen, shortness of breath and palpitation. He was seen by one of us in consultation and then sent into the hospital for further study and treatment.

His past history was significant in that he had had, at the age of eighteen, an attack of "inflammatory rheumatism" in which the muscles of his legs were affected, while his joints remained apparently uninvolved. This illness lasted for four or five weeks, but apparently never recurred. No evidence of any type of venereal disease was elicited.

The pain in the right upper abdomen, which had brought the patient to the hospital, had been present for more than six months. The pain was usually dull but with attacks of sharp stabbing sensations in the same region from time to time. The pains were not related to the taking of food and were not relieved by alkalies He had never vomited. There was a gradual increase in the severity of his pain and within three months he had to give up his work and go to bed. His pain was relieved by rest in bed for five weeks, but a return to work brought on the pain again. No precordial, substernal or radiating pain in the arm had ever been experienced. Some palpitation and moderate shortness of breath had been present from the onset of his illness.

The physical examination revealed no conspicuous abnormalities other than those found in the heart. The cardiac apex impulse was visible and palpable in the fifth interspace 12.5 cm. to the left of the midsternal line. A marked systolic thrill was felt from the aortic area up into the suprasternal notch. A rough aortic systolic murmur, a blowing diastolic murmur and a mitral systolic murmur were heard. The pulse was not definitely anacrotic. The radial artery was moderately sclerosed. The blood pressure was 115/105. There was a secondary anemia. The Wassermann reaction was negative. The urine was normal.

An orthodiagram showed a transversely placed enlarged heart with a lengthened and tortuous aortic outline. The cardiac outline extended 5 cm. to the right and 10.5 cm. to the left of the midline.

A gastrointestinal roentgen-ray examination showed vague evidence of duodenal and appendical pathology.

The diagnosis was rheumatic heart disease with aortic regurgitation and possibly stenosis, beginning cardiac failure and upper abdominal pain as possibly an unusual manifestation of heart disease, possibly of hepatic origin. The probability of an intraabdominal condition, a cholecystitis, a duodenal ulcer or a chronic appendicitis was, of course, not forgotten.

Clinical Course.—During his stay in the hospital the patient suffered at intervals from epigastric distress and had several attacks of severe abdominal pain which required morphine. Alkalies and bland diet gave him some relief. Shortly after

entering the hospital, the patient began to have Adams-Stokes attacks of varying degrees of severity. In the milder attacks, the pulse disappeared for about five seconds, respirations deepened, the face became intensely flushed and consciousness was momentarily lost. In the more severe attacks, the patient was pulseless for twenty seconds or more, dyspnea and cyanosis became marked and clonic convulsions usually occurred. Attacks came in groups, as often as one per minute for fifteen or more minutes, and as many as thirty-four attacks were recorded in one day. During his stay in the hospital he had had one or more attacks on thirty-six days and was free on forty-four days. He was often free from attacks for several days in succession and then had attacks daily for a similar period. In the treatment, epinephrin injections intramuscularly (15 minims of $\frac{1}{1000}$ solution) seemed to relieve the patient on a few occasions. On several occasions atropine hypodermically produced no obvious effect.

Barium chloride was given by mouth in 30 mg. doses thrice daily for thirty-six days with apparently no effect on the frequency of the attacks. This failure, when taken with the early failure of similar therapy in our second case, which later responded so spectacularly, suggests that the fault of the failures was possibly the insufficient dosage. Our best results were accomplished with 50 mg. doses 4 to 6 times per day.

CASE II.—*Adams-Stokes Attacks Occurring During Apparently Complete Heart-Block.*

This case has been previously reported as Case 1 in an article by us in a previous issue of this Journal entitled "Heart Block with and without Convulsive Syncope."[15] The electrocardiographic studies are herein reported in extenso.

The following history notes were abstracted from the previous report:

E. E. M., an American hardware salesman, aged forty-two years, came to the medical clinic of Charity Hospital because of vague symptoms, such as cough, expectoration and slight breathlessness on exertion, which he feared were symptoms of tuberculosis. The patient had not had any of the ordinary clinical symptoms of tuberculosis. He had had no hemoptysis, cardiac pain or edema, but had had some malaise.

In his past history he had had pneumonia and typhoid fever in childhood; malaria at thirty-five years; gonorrhea with a right bubo and a probable neisserian arthritis at twenty-eight. Syphilis was denied. His wives had had no miscarriages and his three children were living and well. His history otherwise yielded no significant facts.

The *physical examination* revealed an aortic facies, pallor, nodding head and throbbing carotids. The heart was enlarged. Loud aortic, diastolic, and systolic murmurs were heard in the second right and the third left interspaces. A systolic murmur was heard at the apex transmitted to the left axilla. The rhythm was regular and the rate was recorded once as 64 per minute. The secondary peripheral vascular phenomena of aortic regurgitation was present. The blood pressure was 168/40.

There was a slight secondary anemia. Otherwise the laboratory tests were negative.

The roentgenogram of the heart showed a cardiac shadow measuring 17.3 cm. in diameter. The aortic hemicircle measured 4.9 cm.

Clinical Course and Treatment.—Potassium iodide in saturated solution in doses of ten drops three times a day and mercury ointment rubs were prescribed. Within two weeks the patient noted an increase in symptoms, especially in shortness of breath. After thirty days of this mixed treatment he noted sudden severe weak spells, fainting sensations and dizziness. The heart rate was 28 per minute. Auric-

ular sounds and irregular intensification of the first sound at the apex were noted as further evidence of complete heart-block.

Barium chloride in 20 mg. doses by mouth was administered every four hours without any apparent effect, even though the first dose given was by mistake sixty times the regular dose. Calcium lactate, 4 gm., was also given every four hours. The pulse remained slow, around 28 per minute, with occasional slight rises even as high as 54. Tincture of digitalis, a total of about 2 c.c., had been administered. The patient fell in fainting attacks twice during his two-day stay in the hospital.

Upon his return to his home he had another attack of syncope. He continued the barium chloride in 20 mg. doses every four hours. The administration of morphine seemed to precipitate a short series of severe attacks. Atropine sulphate hypodermatically was apparently also followed by an extreme exaggeration of the attacks of syncope accompanied by severe convulsions, with a fall in his pulse rate to 16 and once even to 8 per minute. The dosage of barium chloride was increased to 40 mg. every four hours for six doses (240 mg.) per day and epinephrin (0.5 c.c. of $\frac{1}{1000}$ sol.) intramuscularly. Distinct improvement followed within twelve hours. One more convulsive syncope attack occurred, probably precipitated by an overloaded bowel. The barium chloride was increased to 50 mg. doses (300 mg.) per day and continued at this level for a week, after which only three doses of 50 mg. each were given daily for eight weeks and thereafter only one dose per day. During this time the pulse rose as high as 72 per minute and the electrocardiograms described in detail below were taken. The patient was able to return to his work within two weeks after the last attack and has been at work ever since. He continues to take the one 50 mg. dose of barium chloride daily and bismuth salicylate, 0.13 gr. intramuscularly, as an antiluetic drug.

DISCUSSION

Case I. *The Cardiac Mechanism.*—It may be seen by reference to Fig. 1 that 1:1 conduction suddenly gives place to complete block with ventricular standstill. The appearance of complete block is invariably associated with auricular slowing. The electrocardiograms contain the terminations of 18 periods of 1:1 rhythm. The block appears in the different cases when the auricular cycle lengths reach from 0.90 to 1.10 second; the cycles preceding the last transmitted impulse range from 0.62 to 0.98 second. Careful measurement reveals a slight increase in the P-R intervals following the longer auricular cycles. With the onset of ventricular standstill, the auricular rate continues slow for a few cycles and then gradually accelerates, but, as illustrated in Fig. 1, conduction is not thus reestablished. Generally ventricular standstill is interrupted by idioventricular beats arising either below the bifurcation of the bundle of His (Fig. 2, strip 2, complex 2) or above (Fig. 2, strip 2, complex 1). When an auricular systole occurs within an interval extending from 0.31 to 0.795 second after the beginning of the idioventricular beat, conduction invariably follows (Fig. 2, strip 2, P^1; strip 4, P^2). This range is tested 17 times in the curves. Auricular beats falling between the ranges 0.80 to 0.88 are either blocked or transmitted. This range is tested by three impulses, only one of which, following an ectopic beat of nodal

or main bundle origin, is blocked. If the auricular beat comes later than 0.88 second after the idioventricular (Fig. 2, strip 3, P-waves 2 and 4), it is invariably blocked. This is tested by 16 impulses between the ranges 0.88 and 1.28 second, not counting the many additional impulses which are blocked during the period of ventricular quiescence. While the number of impulses with which we deal is relatively low, the chance that these relationships are accidental is extremely remote.

Periods of 1:1 conduction are invariably terminated when the auricular impulse comes later than 0.80 second after the R-wave (Fig. 1, strip 2, P^3). In these cases of 1:1 conduction, if the auricular impulse follows the R by less than 0.685, it is invariably transmitted

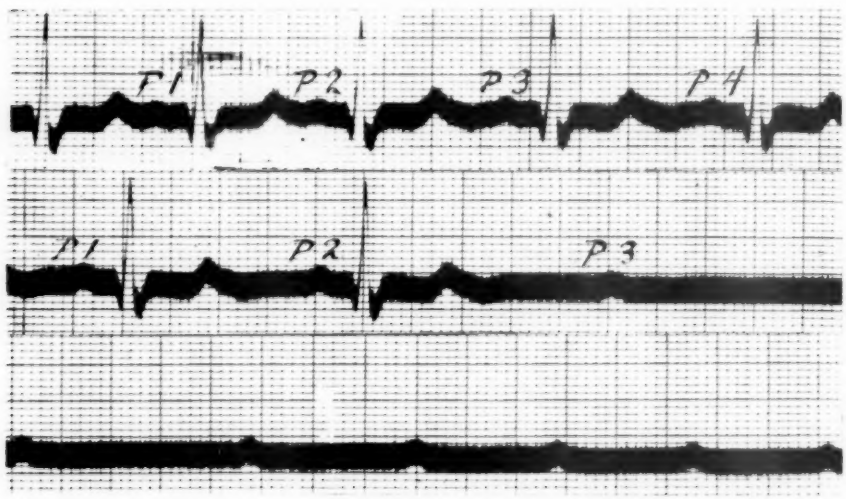

Fig. 1.—Case I, lead 2, continuous tracing. Note the onset of complete block with ventricular standstill when the auricle slows to a rate of about 60 per minute. The ventricular quiescence continued in this instance for at least eighteen seconds when, because of a convulsion, the record was discontinued. The auricular rate rapidly accelerated during the period of ventricular standstill to over 120 per minute. Time in 0.2 and 0.04 seconds.

(Fig. 1, strip 1). If they fall within the range between 0.685 and 0.80 second, they may either be transmitted or blocked (Fig. 1, strip 2, P^2; Fig. 2, strip 1, P^4). Reasons for the different time relations in this case of 1:1 conduction and in the case of auricular impulses following ectopics are given below. When the auricular rate is sufficiently rapid, an idioventricular beat always initiates a period of 1:1 conduction which continues until, with slowing of the auricles, conduction fails (Fig. 2, strip 4). Following the schema adopted by Lewis and Master,[9] Fig. 4 shows the time relations between the P-waves, both transmitted and blocked, and the ventricular complex.

Explanation of the Cardiac Mechanism.—There are three possible causes of the peculiarities of conduction exhibited by this heart.

The *first* is that increased vagus tonus is responsible for the block. The slowing of the auricles is almost certainly due to vagus influence. But two facts make this explanation of the block untenable. (*a*) When the auricles accelerate during a period of ventricular standstill, there is no relief from block until an idioventricular beat occurs. If the vagus were responsible for the block, release of the conducting tissues from vagus influence should occur synchronously with the auricu-

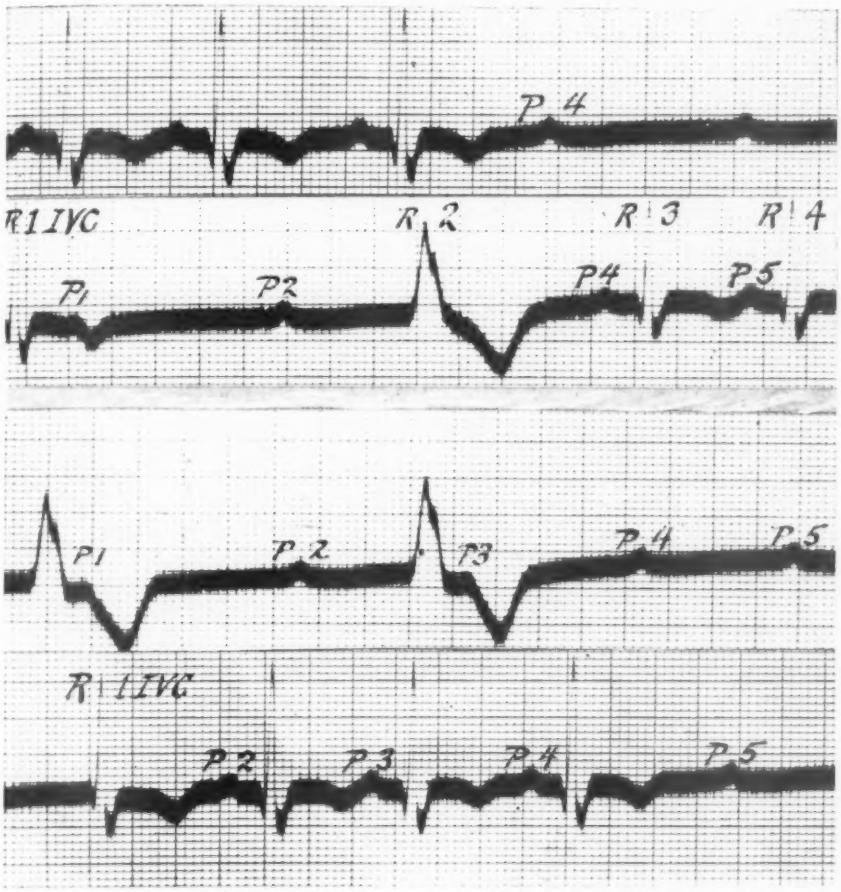

Fig. 2.—Case I, lead 2, first two strips continuous; last two continuous. Illustrates short periods of ventricular standstill interrupted by idioventricular beats of two types with and without subsequent conduction. For details, see text. Time in 0.2 and 0.04 seconds.

lar acceleration. (*b*) As shown clearly in Figs. 2 and 3, transmission is possible when the auricles are slow and when, therefore, the sinus node is strongly under the influence of the vagus.

The *second* explanation is that conduction is in so critical a condition that it fails when slight increases occur in intraventricular pressure. Aortic insufficiency was present and conceivably the re-

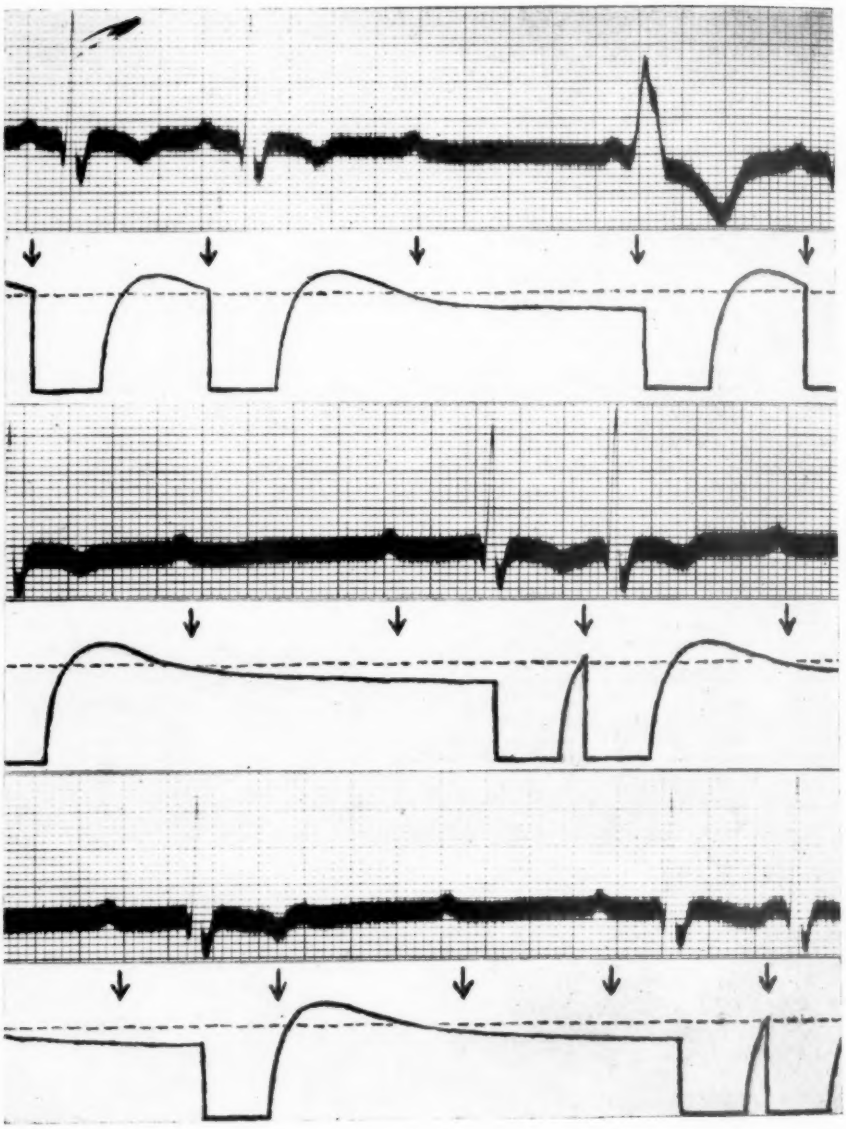

Fig. 3.—Case I, lead 2, continuous tracing. This figure and Fig. 4 illustrate our conception of the rôle of the supernormal phase in producing the peculiarities in conduction displayed by this heart. The first arrow in the diagram below strip 1 represents the instant when the region *C* on the ventricular side of the line of block. *B*, (Fig. 8*A*) is assumed to respond to the auricular impulse. The downstroke of the line of the curve immediately below the arrow represents the onset of the absolutely refractory phase in that region; the horizontal solid line, the assumed duration of the absolutely refractory phase; and the ascending line following, the return of excitability (and conductivity) during the relatively refractory phase. The supernormal crest is reached and, before the second arrow, the beginning of the fall in excitability toward the postsupernormal depression is shown. Before excitability falls below the level of the broken horizontal *threshold* line, a second impulse arrives and another ventricular response ensues. When the third impulse arrives, however, (third arrow) the excitability has fallen below the threshold and the impulse fails to pass the block. The second arrow below the third strip illustrates the arrival of an impulse before the returning excitability has reached the threshold. Since the idioventricular impulses are retrograde (and blocked) the response of region *C* (Fig. 8*A*) is shown as following the beginning of the idioventricular beats. Time in 0.2 and 0.4 seconds.

gurgitating blood raised the pressure sufficiently to produce a mechanical block by the end of about 0.8 or 0.9 second after the beginning of systole. Our discussion of this possibility must come below. It may be stated at this point that Lewis and Master's first case, which was similar to Case I in its most significant features, was not a case of aortic insufficiency.

The *third* interpretation is that recovering conductivity passes through a supernormal phase. Of the three explanations, we strongly incline to the last.

In this first case it is clear that the auricular impulses are blocked during the period of ventricular standstill; they do not reach the ventricle. And it seems equally clear that the retrograde impulses, corresponding to the idioventricular systoles of the two types, are

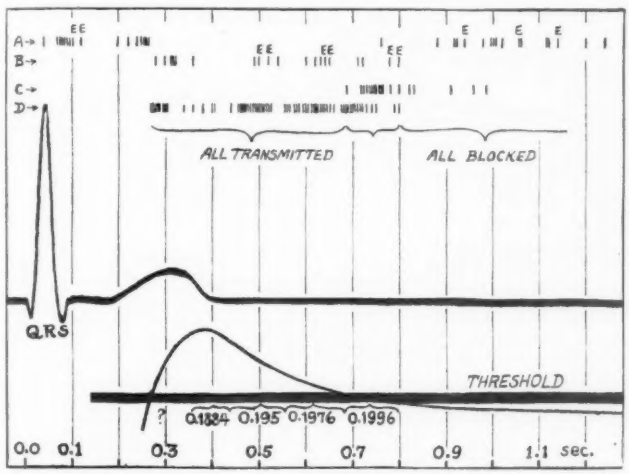

Fig. 4.—Case I. Schematic representation of the time relations between blocked and transmitted P-waves and the R-waves. The row of short vertical lines, *A*, represents blocked P-waves coming, at the time intervals indicated by the numbers below, after previous idioventricular QRS groups. Row *B* shows similarly the transmitted P-waves following the indioventricular beats. Rows *C* and *D* represent, respectively, the blocked (*C*) and transmitted (*D*) P-waves following the ventricular beats of auricular origin. The *E*'s mark the impulses following idioventricular beats arising below the bifurcation of the His bundle. For reasons given in the text and illustrated in Fig. 3 all lines in rows *A* and *B* have been displaced 0.04 second to the left, excepting those marked *E*, which were moved 0.08 second to the left. For further discussion see text.

blocked off from the auricles. We may assume that these impulses are blocked at a line, *B*, shown in Fig. 8-*A*. This is the same diagram previously published by us[15] in explanation of the mechanism of heart-block, excepting that here we include all except the left-hand border of the region, *B*, as illustrated in that paper, as a part of region *C*. *C* represents the injured tissue on the ventricular side of the block, *A* the tissue on the auricular side, *X* and *Y* mark the supposed foci of origin of the two forms of idioventricular systoles.

If we start our discussion with the heart beating coordinately in response to a rapid auricular rhythm, we see that each impulse follows

closely enough after its predecessor to find the tissue of *C* in the supernormal phase. As the auricle becomes slower, each impulse reaches *C* later during the supernormal period and consequently is transmitted slightly more slowly. Finally an impulse follows so late that *C* has become irresponsive, and the impulse is blocked at the line *B*. As long as *C* remains inactive it will stay depressed, and no auricular impulse can be transmitted, no matter how rapid the auricle becomes. These postulated changes in the excitability and conductivity of region *C* are illustrated below the electrocardiograms in Fig. 3. Each transmitted impulse causes the conductivity to fall to zero, as indicated by the vertical lines. This fall is followed by an absolutely refractory period and then a relatively refractory period. The latter begins with the ascent of the line representing excitability and conductivity. A second impulse falling during the absolutely, or very early relatively, refractory period of *C* will find the excitability of *C* below the horizontal line labelled *threshold* and consequently will not be transmitted. Such an impulse is represented by the second P in strip 3 of Fig. 3. The curve then passes through the threshold line. While the curve lies above this line, conduction is possible. Excitability and conductivity reach their highest point at the crest of the supernormal phase, and then there is a gradual depression toward the threshold line, which is crossed unless another auricular impulse arrives before the line is reached. Impulse P^3, strip 3, is blocked because excitability has fallen below the threshold. Other points which must be mentioned are: (*a*) that since the idioventricular impulse will take time after it has been initiated to reach *B* (Fig. 8-*A*), the absolutely refractory period of *C* adjoining *B* is represented in Fig. 3 as beginning shortly after the idioventricular contraction begins, and (*b*) since the transmitted impulse reaches *C* before the ventricular response begins, the absolutely refractory period for these cases is represented as beginning shortly before the R-wave.

We give this schema to represent the mechanism both because it facilitates a visualization of the mechanism as we conceive it, and because it is consistent with the explanation of the mechanism of heart-block which we have previously published.[15] In Fig. 4, following the schema adopted by Lewis and Master,[9] we have illustrated the time relations between the P-waves and the ventricular complexes. (See legend.) The short vertical lines of the row marked *C* represent the blocked P-waves immediately following periods of 1:1 conduction. The lines of the next row (*D*) represent transmitted P-waves during periods of 1:1 conduction; row *B*, transmitted P-waves immediately following idioventricular beats, and row *A*, blocked impulses following ectopics. Below, the curve of recovery of conductivity is drawn. The threshold is represented as a horizontal band. Im-

pulses falling on that part of the curve above the band are transmitted; those below are invariably blocked, and those striking on the parts of the curve within the band are either blocked or transmitted. The reasons for the overlapping of blocked and transmitted impulses at either end of the responsive range are not all clear, but certainly include changes in the condition of the muscle on the auricular side of the block, depending upon the supernormal phase in that region; and possibly nerve influences and fatigue and recovery from fatigue, together with the treppe phenomenon in conductivity. These two latter influences may very greatly influence the supernormal recovery as observed in the bloodless turtle heart.[16] The representation of the recovery curve and the threshold in this manner makes it clear why the overlap of blocked and transmitted impulses is greater at the end than at the beginning of the responsive period.

The numbers below the recovery curve represent the average durations of all impulses transmitted during the time interval indicated. These averages are made from our original measurements and have been modified in no way. The data for the early part of the curve are too scanty to be of any value. We do not care to stress these figures for the average conduction times, since the determinations of the lengths of the P-R intervals were in most cases too uncertain, but so far as they go they are in perfect agreement with our interpretation, and with the form of this part of the supernormal recovery curve as found in the turtle heart.

We may now revert to the second possible cause of the peculiarities in conduction in this heart, namely, that it is attributable to an increase in intraventricular pressure and consequent mechanical block. We believe that the relatively short P-R intervals and the very slight changes in the durations of these intervals, peculiarities shown by most of the previously reported cases of sudden onset of block, count against this view. Our reason for this statement is that similar relations have been observed for the turtle heart, i.e., in some cases all the manifestations of the supernormal phase are observed excepting that the conduction times are short for the apparent degree of compression applied, and the changes in the conduction times associated with the variations in rate are unusually small, although careful measurement generally reveals them. Our second reason for rejecting the mechanical explanation is that block occurs at the close of periods of 1:1 conduction when the R-P intervals reach 0.685 to 0.80 second, while block occurs following an idioventricular contraction only when the R-P intervals reach 0.80 second. This fact is most readily explained on the basis of the supernormal phase, since, as shown above, it is precisely what might have been anticipated. Furthermore, the above-mentioned variations in duration of the P-R intervals are pre-

cisely of the kind which would be expected if the conduction depended upon a supernormal phase, and they are very different from the variations in the spread of the QRS group with varying rest periods which we have described for our second case and which we consider to be due to pressure changes. Our familiarity with the supernormal phase in the cold-blooded heart, under conditions where there can be no question of pressure changes or transient mechanical block, tends to convince us that these latter factors are not responsible for the cardiac mechanism described by Lewis and Master, by Wolferth, and by ourselves.

Case II.—This case, while presenting certain features attributable to a supernormal phase, is also interesting for several other reasons. Perhaps the most remarkable feature is the unprecedented length of the P-R intervals, twenty-nine of those recorded exceeding 0.90 second, while one attains the almost incredible duration of 1.01 second, or nearly seven times the average normal figure. Because of the extreme length of these intervals and the occurrence of ventricular cycles of highly variable duration, the case is an unusually favorable one from which to construct a curve of recovery of conductivity in the injured human auriculoventricular tissues, the first, so far as we are aware, to have been reported. An interesting relationship has also been found to exist between the ventricular cycle lengths, the duration of the QRS complexes and the amplitudes of the chief ventricular deflections. These relationships we have expressed graphically.

The Cardiac Mechanism.—Fig. 5, strip 1, is an electrocardiogram taken on Nov. 27, 1925. All portions of this curve, excepting the one figured, and two other precisely similar portions, presented a picture of typical complete heart-block. All except three of the ventricular cycles are long, averaging 1.35 seconds. The three shorter cycles are each 1.04 seconds in duration. Preceding the R-wave which ends each of these cycles, upon the upstroke of the T-wave of the preceding complex, is a P-wave, and each of these P-waves follows the preceding R by almost exactly 0.24 second. For reasons which will be given in connection with electrocardiograms taken on a later date, we believe that these three P-waves are transmitted. The P-R interval in each case is 0.80 second. The only other curves taken on this date show five additional transmitted impulses, falling within a range extending from 0.08 to 0.40 second after the R-wave. Forty-six P-waves falling within this same range, however, are blocked. Forty-five other P-waves fell later than 0.40 second after the R and yet sufficiently early in diastole to have shortened the ventricular cycle had they been transmitted; yet none caused a ventricular response. Thus, of 54 impulses arising during a responsive phase, 8 were transmitted, while

not one impulse in 45, set up at a time when the conducting tissues had had a *longer* rest, succeeded in reaching the ventricle.

It is only when we compare these findings with those of a later date (Dec. 6) that they assume significance of a convincing nature.* Electrocardiograms taken on this date have already been published in an earlier communication.[15] We present some additional ones in Figs. 5 and 6. Our curves of this date include nearly 600 ventricular

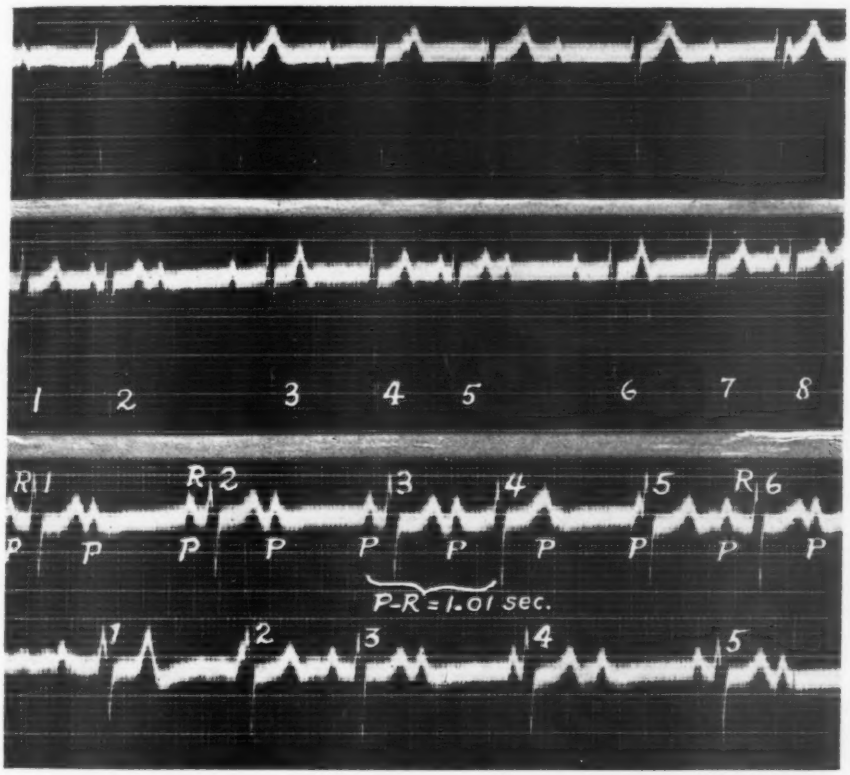

Fig. 5.—Case II, first strip, lead 3, Nov. 27, 1925. Second strip, lead 3, Dec. 6, 1925. In strip 2, ventricular complexes 1, 2, 4, 5, 7, and 8 are in response to auricular impulses. For complexes 2, 5, and 8, conduction time considerably exceeds the auricular cycle length, the transmitted P-waves being lost in the previous ventricular complex. The P-waves following complexes 2 and 5 are clearly blocked. Note also the greater amplitude of the S-waves of 2, 5, and 8, and the intermediate amplitude of 1, 4 and 7.

Third and fourth strips, lead 2, continuous tracing. Similar to the second strip but not regularly repeated groups of ventricular beats. Complexes 2 and 3, strip 3, and 1, 4 and 5, strip 4, are idioventricular. The rest are responses to auricular impulses. The P-R interval marked is the longest ever recorded, approximately 1.01 seconds. Time in 0.2 and 0.04 second.

complexes, of which not quite 200 are responses to auricular impulses. Reference to the figures just mentioned and to Figs. 5 and 6 will make clear the salient features of the cardiac mechanism.

*Electrocardiograms similar to these of Dec. 6 were also obtained on another day, but were unfortunately so poor as to make accurate measurement impossible. They showed nothing inconsistent with our interpretations, however.

Fig. 7 is constructed in the same way as Fig. 4 (Case I), except that the transmitted impulses are represented by heavy dots instead of lines, and the conduction time for each impulse is indicated by its position with reference to the figures along the ordinate. As in the previous figure, the position of the dot along the abscissa fixes the time relations of the beginning of the P-wave with reference to the ventricular complex. It will be observed that no P-waves are transmitted which fall earlier than 0.18 second before the beginning of the R-wave. As the P-waves approach closer and closer to the R, their chance of transmission becomes progressively better until, during an interval extending from 0.08 before to 0.09 second after the R, the great majority are transmitted. Between 0.09 and 0.30 second

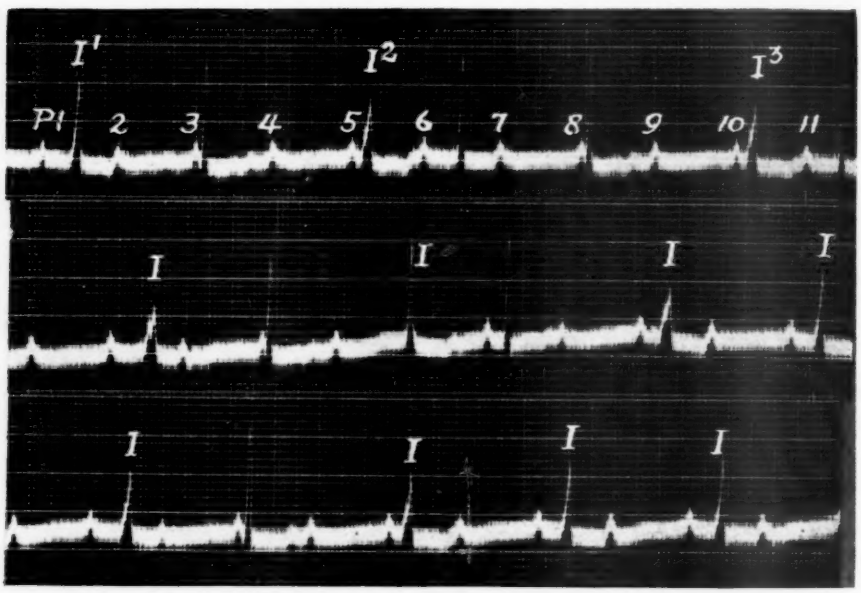

Fig. 6.—Case II, lead 1. Continuous tracing. Complexes marked *I* are idioventricular; the remainder are responses to auricular impulses. Note that P-waves 5 and 10 in strip 1, despite their occurrence just before an idioventricular complex of abnormal form, are transmitted (see last section of paper). Note, in all the electrocardiograms from Case II on this date, the variable time relations between the idioventricular beats and the preceding P-waves, an argument that they are idioventricular; and note also that no ventricular beat follows a preceding one by less than 1.20 second unless also preceded by an appropriately located P-wave. All P-waves located after the T, like the ninth in strip 1, are blocked. Time in 0.2 and 0.04 second.

after the R, all impulses reach the ventricle. Beyond 0.30 second blocked P-waves become more and more frequent until, beyond 0.40 or 0.45 second all impulses are again blocked. We cannot test the range beyond about 0.65 or 0.70 second, since, if transmitted, the response of the ventricle to the auricular impulse would be anticipated by an idioventricular beat.

Since our whole interpretation of this case depends upon our con-

viction that the majority, if not all, of the impulses indicated by the black dots in Fig. 7 are transmitted, it becomes imperative to present our evidence for conduction. (*a*) The most cogent evidence for conduction is the invariable failure of ventricular systoles to appear within an interval of less than 1.20 seconds after a previous ventricular systole unless preceded, at a proper time interval, by an auricular systole. (*b*) Our second argument is an outgrowth of the first. When con-

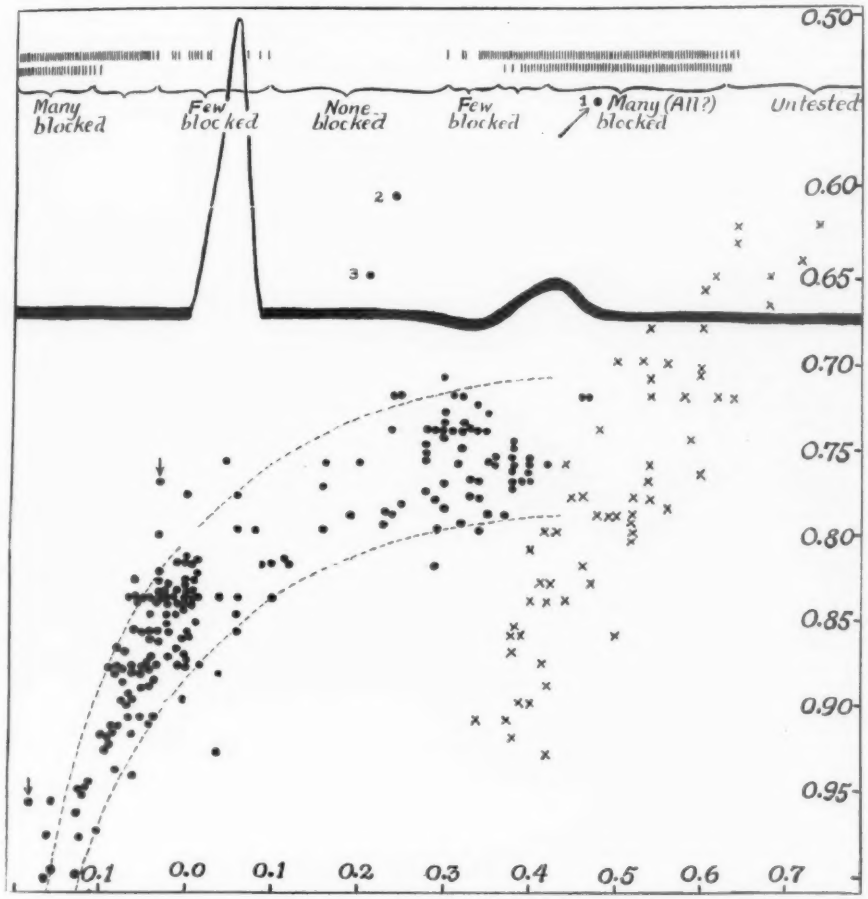

Fig. 7.—Case II. Dots represent transmitted P-waves. The position of each dot with reference to the ordinate gives the P-R interval; its position along the abscissa, its time relations with the nearest ventricular complex, which is shown above the curve. All numbers are seconds. The *X*'s represent P-waves preceding ventricular complexes terminating cycles of from 1.20 to about 1.34 seconds. The significance of the scattering of the *X*'s is discussed in the text. The marked deviation of points 1, 2 and 3 was apparently due to marked auricular slowing with probable cumulative recovery from fatigue.

The double row of short vertical lines at the top represents the positions of the blocked P-waves. No attempt was made to include all of them excepting in the range extending from 0.10 before to 0.38 second after the beginning of the R-wave.

duction times are plotted as ordinates, and the time relation of the P-waves with reference to the R-wave as abscissae, we obtain the curve illustrated in Fig. 7. As it stands, this curve cannot be acci-

dental, and, as we shall show, when corrected for variations in auricular rate, the points along the curve are less scattered. When it is further considered that the curve is of the form which has been found to be characteristic of the recovery of conductivity in animal hearts under experimental conditions, we believe that the evidence for conduction becomes as strong as it is possible to make it.

The Supernormal Phase.—Of the criteria which we regard as indicative of supernormal conduction in the heart, this case presents but one, i.e., the transmission of impulses during a portion of the cycle when the tissues have had a relatively short rest, their blocking after a longer rest interval. The other chief criterion, the more rapid transmission of impulses after a relatively short rest and their slower transmission later, is clearly not shown. In fact the impulses, as Fig. 7 demonstrates, are transmitted more and more rapidly with longer rest intervals until, with further extension of the rest period, they are blocked. This does not, however, make it necessary to abandon the supernormal phase in our interpretation. We have previously[15] proposed the view that the prolonged conduction time in this heart is due to the extent of the injury; that the impulse must travel for a longer distance through injured muscle than in the usual case of heart-block. Now, the peculiarities of conduction in this heart are understandable if it is assumed (*a*) that a comparatively short stretch of the conducting tissue is more severely injured and at a relatively high H-ion concentration, so that the conditions obtain which favor the appearance of the supernormal phase in recovery, and (*b*) that the rest of the injured tissue recovers along the usual curve for injured muscle. The short stretch of more injured tissue could manifest all the phenomena characteristic of the supernormal phase, but the changes in conduction time in this region, if of the same order of magnitude as in Case I, would have no demonstrable influence upon conduction time as a whole.

The Recovery Curve of Conductivity.—In Fig. 7, as has already been noted, is given the curve obtained when the P-R intervals are plotted against the position of the transmitted P with reference to the R-wave. Inspection reveals considerable scattering of points so that an accurate determination of the form of the curve is impossible. We believe that there are at least two reasons for the scattering: (1) changes in conduction time due to variations in auricular rate, and (2) incorrect representation of the true duration of the rest intervals on the ventricular side of the actual point of block. Before presenting a curve in which we have corrected for these influences, it is necessary to explain more in detail our conception of the actual cardiac mechanism and the method by which correction was made. Fig. 8-*B* shows the auricles, ventricles, and a stretch of injured muscle between

them. *B* is the line at which the blocked impulses are regarded as stopping; *A*, the injured muscle on the auricular side of the block; *C*, the injured muscle on the ventricular side. Now, if we suppose that the idioventricular beat I^2 in Fig. 6 arose, as its form might indicate, below the bifurcation of the His bundle, we would experience difficulty in accounting for the transmission of the auricular impulse, P^5. We might, of course, assume that the block is very close to the ventricle and that almost all the delay in conduction occurs on the auricular side of the block. Thus impulse P^5 may be transmitted, because, by the time the impulse reaches the block, the tissue on the ventricular side of the block will be ready to respond. But such an explanation will not account for the further fact that the greater part of the variation in the durations of the P-R intervals (0.65 to 1.01 sec.) is due to the varying durations of the rest intervals on the ventricular side of the block. In order that such variations may occur we must believe that all, or certainly most of the idioventricular impulses arise not far from the block, e.g., at the point marked *x* in Fig. 8-*B*. We have given independent evidence for this assumption below (last section).

It will be apparent, therefore, that the idioventricular impulse will arise some time before it appears on the electrocardiogram. But by the time the auricular impulse set up 0.1 second or more before the appearance of the idioventricular beat has reached the block, the region *C* (Fig. 8-*B*) will have recovered sufficiently to make transmission possible.

It seems impossible to determine with certainty what proportion of the total conduction time is lost respectively in regions *A* and *C*. It is possible, however, empirically to draw separate recovery curves for regions *A* and *C*, and, knowing the duration of the auricular cycle preceding conduction, to calculate the relative duration of the rest period in *C* at the time each impulse arrives from above. From these figures the whole P-R interval can be calculated. In Fig. 8 the true conduction time, as measured on the electrocardiogram, was corrected for the duration of the preceding auricular systole, i.e., for the rest interval in region *A*. These corrected intervals, as ordinates, are plotted against the calculated rest periods in region *C* as abscissae. The scattering of the points, therefore, indicates the extent by which the true conduction times deviate from the calculated intervals. The results are not wholly satisfactory, but the scattering is much less pronounced than in the uncorrected curve of Fig. 7, and we believe the corrected curve is a fairly close approximation to the truth. Unavoidable error in the measurement of the P-R intervals, cumulative fatigue and rest effects with varying auricular rates and with sequential transmissions of impulses, nerve influences in region *A*, and

perhaps failure to find the best possible empirical curves as a basis for calculation may all be responsible for this deviation of the points. The points marked 1, 2, 3 deviate from the calculated curve presumably because with marked auricular slowing there was some recovery from cumulative fatigue. The points marked by the arrows were very possibly ectopic beats and if so do not represent true conduction. These points are similarly indicated in Fig. 7.

One fact is brought out in a striking fashion by this treatment of our data. The points corresponding to P-R intervals terminating R-R

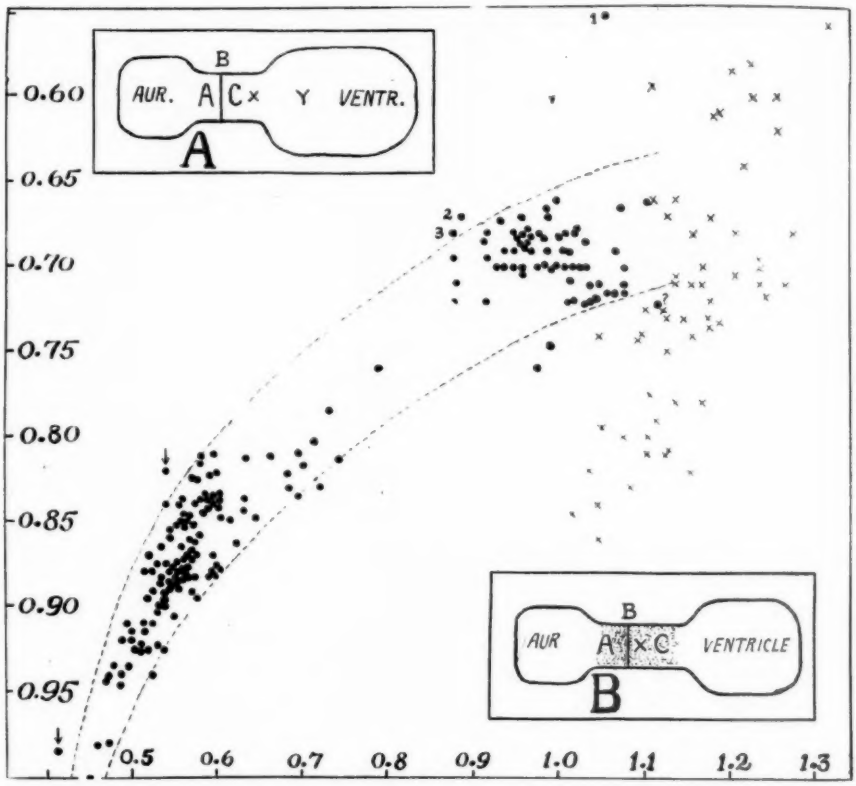

Fig. 8.—*A*.—Diagram referred to in text in discussion of Case I. *B*. Similar to that shown in Fig. 7, excepting that the P-R intervals, shown by the position of the dot with reference to the ordinate, are corrected for the auricular cycle length preceding the transmitted P; and the rest intervals, shown along the abscisse, are calculated. The abscissae represent the calculated rest intervals at the left hand border of region *C*, the shaded area on the right side of the line of block, *B*, in Fig. 8*B*. These rest intervals are not absolute, but relative only. The numbered points and those shown by the arrows are the same as those similarly indicated in Fig. 7. The *X*'s also correspond to the *X*'s in Fig. 7. As in the previous figure, all recorded conduction times of December 6 are included.

intervals of 1.20 seconds and above have a random scattering. These points for all R-R intervals of 1.20 to 1.34 seconds are represented by the *x*'s in the figure. This we believe is almost conclusive evidence that R-R intervals (ventricular cycles) of above 1.20 seconds were always, or nearly always, terminated by an idioventricular beat. In the same

way, the absence of extreme scattering for points corresponding to P-R intervals ending ventricular cycles of shorter length is equally good evidence that most, if not all, of these shorter intervals were brought to an end by a ventricular response to a transmitted impulse.

The form of this curve of recovery of conductivity is in close agreement with those found by Ashman[17] for the compressed cardiac muscle of the turtle heart, and by Lewis and Master[18] for the uninjured conducting tissues of the dog's heart. Ashman found that compression of the tissues not only increased the conduction time, but greatly lengthened the time required for the return of conductivity to its resting level. This human curve exemplifies the same phenomenon. Conduction is not back to a resting level at the end of a second or more. In the normal heart of the dog, according to Lewis and Master, recovery is complete in approximately 0.3 second, and certainly in the normal human heart the recovery must be practically complete in 0.5 second or less.

The Prolonged P-R Intervals.—These have been mentioned in our previous paper.[15] In that paper, however, we did not give the evidence proving conduction in case of our longest intervals. Such evidence has already been presented in this communication. The longest P-R intervals hitherto reported on the basis of electrocardiographic evidence are those in a case of Thayer's.[19] His published curves show P-R intervals averaging 0.68 second, and one interval, not, however, certainly representing conduction, of 0.88. The average conduction time in our second case, on Dec. 6, was 0.83 to 0.84 second. Only three intervals were shorter than 0.70 second, while 40 P-R intervals exceeded 0.88 second; 11 were 0.95 or longer; one was 1.00 and another 1.01 seconds (Figs. 5 and 7).

The Relation Between Ventricular Cycle Length, the Duration of the QRS and the Amplitude of the Chief Ventricular Deflection.—One of the striking features of the electrocardiograms of Dec. 6 is the decrease in the amplitude of the chief deflection of the QRS group and the increase in its duration with long ventricular rest intervals. We at first attributed the broad low form of the QRS to an origin below the bifurcation of the His bundle, but, as stated above, we soon found that if such an assumption were made it was impossible to account for the conduction of certain auricular impulses. We then measured the amplitudes of the chief deflection and the spread of the QRS. The curves in Fig. 9 illustrate the result and, we believe, bear out our conclusion that most, if not all, of the idioventricular beats have their origin well above the bifurcation. For, if we assume that the idioventricular beats terminating the longest R-R intervals have their origin below the bifurcation, then it is necessary to assume that as the idioventricular rate becomes more rapid the pacemaker shifts

more or less gradually toward the bifurcation. This would perhaps not be an improbable assumption were it not for the fact that the idioventricular curve is perfectly continuous with the curve for the transmitted impulses. Thus the points at 1.00 to 1.20 seconds along the abscissae, which are responses to auricular impulses, already indicate a slight widening of the QRS and decrease in amplitude of the chief deflection. If the change in the QRS were due merely to point of origin, then we should expect all points representing ventricular responses to auricular impulses to lie along a straight horizontal line, and a definite break to appear at 1.20 seconds between the first and second portions of the curve.

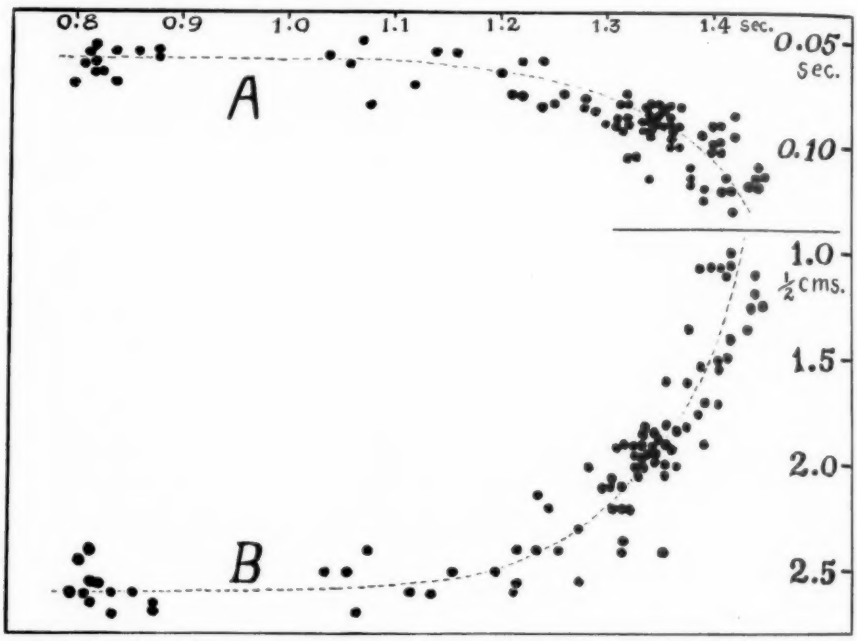

Fig. 9.—Case II. *A*. Curve constructed by plotting the durations of the ventricular cycles as abscissae against the durations of the QRS groups as ordinates. The longer the ventricular rest, the wider the QRS (see Fig. 6.) *B*. Abscissae as in curve *A*; ordinates, the amplitudes of the R-waves in half-centimeters. Both these curves are from Lead I. Curves from the other electrocardiograms are in full agreement with these (see Fig. 5, strip 2). Discussion in text, last section.

An interpretation of this phenomenon is not easy, beyond the evident fact that there is aberration in the spread of the impulse through the ventricle. It seems improbable, in view of our present knowledge of the supernormal phase, that it is to be explained on that basis in spite of the fact that superficially there is a resemblance, i.e., the longer the rest interval the slower the conduction through the ventricular muscle. If the curve *A*, for example, represented the onset of the post supernormal depression, we should expect the curve to be concave on its upper side, not convex.

If we reject the supernormal phase as a probable factor, there re-

mains increase in intraventricular pressure with consequent mechanical block as an alternative explanation. This, it will be recalled, is precisely the factor which we were disinclined to regard as the cause of the phenomena which we attributed to a supernormal phase in Case I. And we believe that in this case attribution of the phenomenon we are discussing to pressure changes does not weaken our argument for the supernormal phase in A-V conduction. In order to make our argument clear, we must refer once more to Fig. 8-*B*, and consider the passage through the injured A-V tissue of an auricular impulse which arises just early enough to escape block. The average P-R interval for such an impulse is about 0.74 second. Let us suppose that 0.37 second, or half the time, is lost in region *A*; and 0.37 in region *C*. Then the impulse will be leaving *B* about 0.82 second after the previous ventricular systole, or at a time when, according to Fig. 9, no detectable change in conductivity has occurred in the ventricular muscle. By the time it reaches the ventricles, however, e.g., 1.18 seconds after the previous systole, and the ventricular response occurs, a slight depression in conductivity has appeared, as shown by the curves. Now an impulse arising only 0.02 or 0.03 second later in the auricles is likely to be blocked. Yet when it reaches *B* (Fig. 8-*B*) conditions, so far as pressure is concerned, are not very different from those met by the other impulse we have considered. If the impulse is blocked by *pressure*, we should expect it to occur later, after the impulse has passed *B*, and has reached some point lower than *x*. Yet no complete block is present at that time or even much later in that region (*C*), since the idioventricular impulses, which we believe must arise at *x* close to *B* are not blocked in their passage from *x* to the ventricle. We must, therefore, believe, if our assumptions are right, that the impulse is blocked before it reaches *C*, and that the cause of block is a postsupernormal depression rather than pressure.

This argument, as we realize, is not conclusive, and the presence of a true supernormal phase in our Case II must remain somewhat in doubt. If, however, it should be shown not to exist, the demonstration would not in any way detract from the interest of the three other phenomena to which we have directed attention.

SUMMARY

1. Two cases of A-V heart-block are reported which we believe illustrate the supernormal phase in conduction.

2. Our second case, in which the argument for the supernormal is less strong, is of additional interest because of (*a*) the unprecedented length of the P-R intervals, which reach 1.01 seconds; (*b*) the variation in the duration of the rest intervals and of the P-R intervals, permitting the graphic representation of a recovery curve of conduc-

tivity, and (*c*) the spreading of the QRS group and the decrease in the amplitude of the chief ventricular deflection with longer ventricular rest periods.

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PROGNOSIS IN RHEUMATIC, HYPERTENSIVE AND SYPHILITIC HEART DISEASE*

EMMET F. HORINE, M.D.
LOUISVILLE, KY.

IN THE past prognosis has been largely speculative, but now with increasing knowledge it rests upon a fairly firm foundation. In tracing the development of prognosis in cardiology it is interesting to review the ideas held by William Stokes[1] almost a century ago. He called attention to the fallacy of the opinion then generally entertained as to the great liability to sudden death in all forms of valvular defect. He was a pioneer in attempting to show that the prognosis should be based more on the condition of the myocardium than on the defect itself.

A study of approximately three thousand miscellaneous heart cases has been made but impressions will be given concerning the following types only: (1) rheumatic heart disease; (2) hypertensive heart disease; (3) syphilitic heart disease. Percentages will not be given for the reason that in so relatively small a number of cases such a method savors of a certainty which does not exist.

At the outset it is well to inquire as to what is the foundation upon which a prognosis should rest. With four factors carefully worked out a prognosis may be deduced which will prove fairly accurate. The essential factors are (1) history; (2) etiology (active or quiescent); (3) structural changes; (4) instrumental data: (sphygmomanometer, sphygmograph and electrocardiograph). Finally the response to treatment is of value in further amplifying the prognosis. In my experience neither vital capacity measurements, the various exercise tolerance tests, nor the Schtange test have been of much aid in arriving at a prognosis.

RHEUMATIC HEART DISEASE

With a rheumatic etiology, with either early mitral stenosis or aortic insufficiency present, with no recent activity, with any probable focus for reinfection removed, and with proper supervision, such a cardiac patient may look forward to many years of useful life. Particularly is the prognosis good if many years have elapsed since the original infection and if no evidence of congestive failure has ever been manifest. In fact, barring some severe intercurrent infection or reinfection, this type of patient may even live out his expectancy. The picture presented above, however, is not often met with in its entirety

*Read before the American Heart Association, Dallas, Texas, April 20, 1926.

and the prognosis must be modified accordingly. When periodic health examinations become more generally popular such cases will be found more frequently.

In rheumatic hearts with a history of frequent periods of congestive failure or with evidence of continued active infection there is no real hope. Further if, with careful treatment for months, evidence of failure persists, the outlook is bad, even though the rhythm remains regular.

Auricular fibrillation or flutter, whether paroxysmal or permanent, adds somewhat to the gravity of the situation. If the ventricular rate can be held in check by digitalis, however, the patient may go along in comfort for years. With auricular fibrillation present it is well to advise the family that such a complication does carry with it the possibility of sudden death either from embolism or ventricular fibrillation.

The consensus of opinion seems to be that in rheumatic heart disease the prognosis is somewhat better in women than in men. My own observations are in accord.

HYPERTENSIVE HEART DISEASE

Hypertensive heart disease is by far the most prevalent type encountered among adults after the fortieth year. But even taking in all age groups I have found it to occur almost as often as all other lesions combined. In my own work, of physicians who have been examined and found to have some real heart lesion, 85 per cent have had hypertensive cardiovascular disease.

When manifest evidence of congestive or anginal failure arises in hypertensive heart disease it is the beginning of the end. From two to five years is the average duration of life when the condition is discovered so late. If the condition be discovered prior to the onset of symptoms then the prognosis is much better, for my experience teaches me it is amenable to treatment and may be held in abeyance for years. If periodic health examinations were made for no other purpose than the discovery of early hypertension they would be of utmost importance. Hypertension occurring under thirty years of age seems less amenable to treatment than when occurring later in life.

In hypertensive heart disease instrumental evidence is of inestimable value in arriving at a correct prognosis. In so far as prognosis is concerned the relative value of the various instruments is as follows: (1) electrocardiograph; (2) sphygmograph; (3) sphygmomanometer.

The electrocardiograph is of vital importance in studying hypertensive cardiovascular disease from a prognostic point of view. Vary-

ing grades of heart-block, peak-notching of the QRS group, low voltage of the QRS group, inverted T-waves, coronary T-waves, bundle-branch blocks, all shown electrocardiographically, constitute important evidence. Willius,[2] Pardee,[3] Hay[4] and others have made valuable contributions along these lines.

Intrinsic heart-block may be present in hypertensive disease and yet the individual may be able to carry on for years. Its presence indicates definite myocardial change and the prognosis is influenced accordingly.

Either peak-notching of the QRS or low voltage of the QRS occurring in hypertensive heart disease renders the prognosis graver than otherwise. Inversion of T, not dependent on some drug (digitalis, quinidine, morphine, etc.), is not a favorable finding. A negative T in Lead I alone, in my experience, seems to be less grave than when the T is inverted in all leads, in both Leads I and II or in both II and III. A negative T in Lead III alone has no prognostic significance. The prognosis is especially grave in patients showing an inverted T in all three leads and in addition an upward convexity of the S-T interval in all three leads. No one in whom I have found the last mentioned combination has lived longer than one year and the majority have died within a few months.

Pardee[3] has called attention to the *coronary T:* an upward convexity followed by a sharp downward peak in one lead. He states that if this occurs in Lead III alone it is not significant unless the T of Lead II is inverted, although the upward convexity need not be present in II. This is believed by Pardee to be "characteristic of coronary artery occlusion with an area of deficient blood supply in the ventricular muscle." Those in whom I have discovered this abnormality, even though no urgent symptoms were present at the time, have quite speedily developed grave symptoms and succumbed in less than two years.

Bundle-branch block is an exceedingly grave finding in hypertensive heart disease. Those in whom electrocardiographic evidence of this lesion has been a constant finding have died within eighteen months or less.

As in rheumatic heart disease the supervention of either auricular flutter or fibrillation adds to the gravity of the prognosis. The response to treatment may at times be quite good, in which event the chronic fibrillator may go for several years in comfort.

Wenckebach[5] is of the opinion that sinus arrhythmia may be considered as a favorable omen. Mackenzie[6] once made the statement that he had never seen sinus arrhythmia occur with a "progressive lesion of the heart muscle." He suggested that this would be a good subject to follow up and this I have attempted to do for a period

of eight years. It has been my fortune to run across many patients showing progressive lesions, not only in hypertension cases but also in the rheumatic group, with sinus arrhythmia. McCrudden[7] also recently reported one hundred cases showing definite rheumatic involvement, and in eighty sinus arrhythmia was present. Therefore, I do not feel that because sinus arrhythmia happens to be present in a heart case that we can consider the patient as not having a progressive lesion. The presence of sinus arrhythmia is merely the expression of variable vagal inhibition, while its absence indicates heightened sympathetic effect. In neither event does it speak either for or against the integrity of the myocardium.

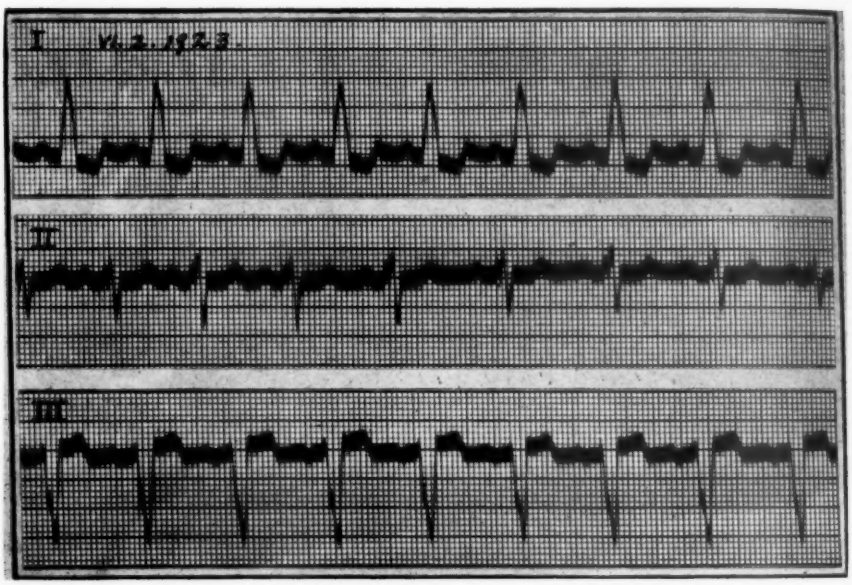

Fig. 1.—Record of combined right bundle-branch block (indicating rather widespread myocardial changes) and sinus arrhythmia. Female, aged fifty-nine years with hypertensive heart disease and presenting decided congestive failure. Death occurred twenty-seven months after record was obtained.

The presence of pulsus alternans in hypertensive heart disease is a definitely bad prognostic sign. Whether the finding is constant or occurs only after premature beats does not seem materially to alter its significance. The studies of Mackenzie,[6] of Paul D. White[8] and of Windle[9] all point to the gravity of this finding.

In so far as blood pressure readings are concerned, Norris[10] has called attention to the seriousness of high diastolic pressures. While high diastolic levels carry with them a high ultimate death rate, they do not necessarily indicate an early death unless constantly maintained at 150 mm. Hg. or above.

SYPHILITIC CARDIOVASCULAR DISEASE

Since the earlier stages of cardiovascular syphilis are symptomless, it is only the relatively advanced case that usually consults the physician. Even though additional structural changes may possibly be halted, the individual still has manifest defects with which to cope. For example, despite halting the progress of a syphilitic aortic insufficiency, such a lesion carries with it a far graver prognosis than a similar quiescent rheumatic aortic insufficiency. The explanation for this difference is readily found when we study the pathology of the two lesions. With syphilitic involvement of the aortic ring there will likewise be found, as a part of the aortitis, a narrowing of the coro-

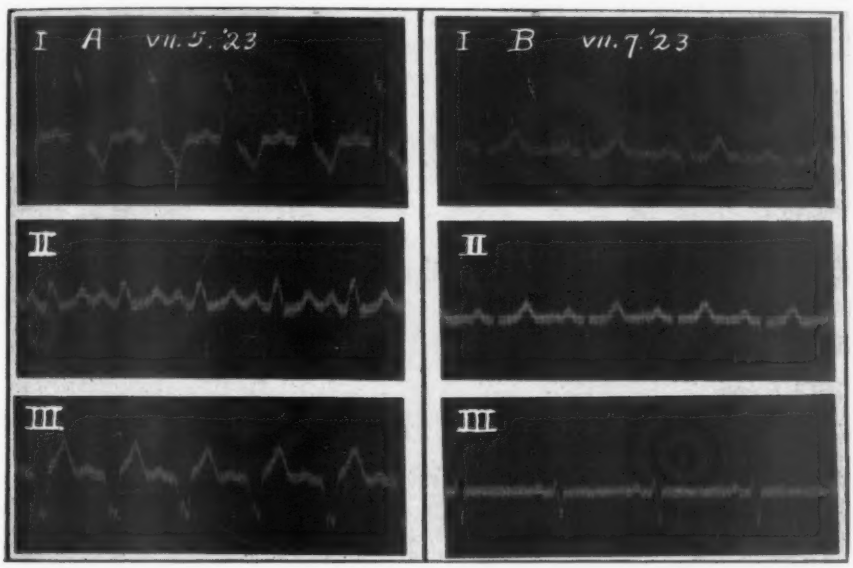

Fig. 2.—Record *A* suggests right bundle-branch block. This occurred as a paroxysmal condition, being accompanied by substernal oppression and pain in the left arm and hand. *B* was obtained two days later during an interval of freedom from pain. Female, aged forty-six years, with syphilitic heart disease decidedly improved after three years' treatment with mercury and the iodides.

nary lumina. Actually there is an infiltration of the intima of the coronaries for a distance of from a half to even as much as two centimeters from the ostia, the remaining portion of the vessels showing less involvement. Frequently, at autopsy, I have found the mouths of the coronaries almost closed. Such a narrowing of the coronary lumina must result in undernourishment of the myocardium. In the uncomplicated rheumatic aortic insufficiency, the openings into the coronaries are normal, the heart having to cope only with the added strain of the insufficiency which alone is not very great.

In cardiovascular syphilis, death usually comes within three years or less time from the beginning of manifest symptoms. Particularly

is the prognosis grave if congestive failure be present when the patient is first seen, death usually ensuing within less than a year. There will be an occasional exception to these general statements, as evidenced by a patient whom I have under observation at the present time. The patient, a man aged seventy-four years, came with an aortic insufficiency twelve years ago following an initial lesion eighteen years previously. His blood Wassermann was positive (+++). The arch of the aorta showed definite widening, having a transverse diameter of 9.2 cm. The heart showed a transverse diameter of 17.8 cm., the internal transverse diameter of the chest being 30 cm. There was no history of any one of the rheumatic group of infections, so that with the picture presented there can be little doubt that the aortic widening and aortic insufficiency were both syphilitic in origin. He has been given courses of mercury and the iodides each year since then with intermissions of six weeks. At present he is showing moderate congestive failure, so that the end cannot now be long delayed.

Patients presenting syphilitic auriculoventricular block or intraventricular block have, if the block was constant, died within a year. One patient presenting paroxysmal intraventricular block of the right bundle-branch type but without aortic insufficiency is still in good condition after three years. The treatment has been courses of mercury and iodides with six weeks intermission annually.

CONCLUSIONS

This necessarily brief survey of the prognosis of the three larger groups of heart disease reveals to us how powerless we are in the later stages of the disease. We see, too, the possibility of doing considerable good when the patients are seen sufficiently early. The entire situation hinges upon the necessity for periodic health examinations which will reveal heart disease in its earlier stages or even in its potential state. As cardiologists, then, let us aid in every possible way to spread the gospel of the periodic health examination and, by so doing, make possible a brighter prognosis.

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(For discussion see page 649.)

THE PROGNOSTIC SIGNIFICANCE OF SEVERAL COMMON ELECTROCARDIOGRAPHIC ABNORMALITIES*

J. Hepburn, M.B. (Tor.), and R. A. Jamieson, B.A., M.B. (Tor.)
Toronto, Canada

EARLY studies in electrocardiography indicated that its chief usefulness in clinical medicine would lie in the differentiation of the various cardiac arrhythmias. While this was of inestimable value, still, once it had cleared up the mystery clouding the production of many of the cardiac irregularities, it told little, in the average patient, that could not be ascertained by careful clinical investigation. It appeared at first as though the electrocardiogram was to be of little or no prognostic value in the study of cardiac disease. The later work of Eppinger and Rothberger[1] and Lewis,[2] however, suggested that certain abnormal types of the electrocardiogram were associated with changes in the heart muscle, indicating that the electrocardiogram might be of aid in prognosis. More recent investigations by Carter,[3] Oppenheimer and Rothschild,[4] Smith,[5] Willius,[6] Pardee,[7] and Cowan and Bramwell[8] have confirmed these observations. In this investigation an attempt has been made to demonstrate the significance and value in prognosis of certain changes in the electrocardiogram in patients suffering from myocardial disease.

The majority of the patients studied have been under observation for many months, and fairly frequent clinical observations have been made. The present report, however, is not primarily a clinical one, but rather an investigation of certain electrocardiographic abnormalities, and an endeavor to ascertain any bearing these abnormalities might have on the duration of life. With very few exceptions all the patients investigated presented symptoms of cardiovascular disease.

The electrocardiographic abnormalities studied and presented in this series are negative T-waves, bundle-branch block, auricular fibrillation with ventricular extrasystoles, and low voltage. These are classified and grouped according to the abnormal electrocardiographic conditions found. The statistics are all based upon the cases traced, and among fatal cases only cardiac deaths are included. Patients still alive, who had been under observation for less than six months, have been omitted from the series. As it was impossible to determine the actual time of onset of any of the abnormal conditions studied, wherever length of life is mentioned it denotes the time elapsing

*From the Department of Medicine, University of Toronto and Toronto General Hospital.

since the patient first came under observation. The average duration of life of patients who have died, and also of those still alive is given. The latter would seem of value in that, in all instances, the average duration of life of those patients still living exceeds definitely the duration of life of the patients who have died. It would appear, therefore, that the consideration of the mortality statistics alone gives too grave a prognosis. The average age of the dead and of the living is included in the tables in order to see whether age might be a factor in the result. A minute etiological subdivision has not been followed because a particular disease might only claim one or two cases and this appeared too small a group upon which to base conclusions.

NEGATIVE T-WAVES

Patients showing low voltage, bundle-branch block, and all patients who received digitalis within two weeks prior to the examination have been excluded from the negative T-wave groups. Negative T-waves occurring in Lead III alone have not been included, these variations being considered of no significance.

Negative T-waves in Lead I.—In this group 40 patients have been traced, of whom 26 had arteriosclerosis or hypertension. The remaining 14 were about equally divided among rheumatic heart disease, syphilis, hyperthyroidism and bacterial endocarditis. Forty-five per cent of the patients of this group are dead. The time which elapsed between first examination and death varied from less than 1 month to 39 months, the average being 11 months. Of these patients 55 per cent are still alive, the average length of life being 12 months, the maximum 36 months.

Negative T-waves in Leads I and II.—Ten of the 20 patients in this group had arteriosclerosis or hypertension. The remainder were about equally divided between rheumatic heart disease, syphilis and hyperthyroidism. Fifty-five per cent of those traced are dead, the average length of life being 5.8 months. The variation in life ranged from less than 1 month to 20 months. Forty-five per cent of the patients are still living, the average duration of life being 18.6 months and the maximum to date 47 months.

Negative T-waves in Leads II and III.—In this group 24 patients have been traced, of whom 11 had arteriosclerosis or hypertension; 7 had rheumatic heart disease; 2 had syphilis; 1 bacterial endocarditis; and 3 were clinically noncardiac. Of the patients in this group 50 per cent are dead, the average length of life being 9.7 months, and the variation in length of life ranging from less than 1 month to 37 months; 50 per cent are still alive, with an average length of life of 17.5 months, the maximum duration being 49 months.

Negative T-waves in Leads I, II and III.—Thirteen patients in all have been traced; 6 of these had rheumatic heart disease; 4 had arteriosclerosis; and 3 had syphilis. Of these patients 46.1 per cent are known to be dead, the average length of life being 6.6 months, and the extreme range from a few weeks to 33 months; 53.8 per cent are still alive, with an average duration of life of 15.3 months and a maximum of 45 months.

TABLE I

	Cases heard from	PATIENTS DEAD			PATIENTS LIVING				
		No. of cases	Percentage	Average length of life in months	No. of cases	Percentage	Average length of life in months	Average age of dead in years	Average age of living in years
T_1 negative	40	18	45	11	22	55	12	52.7	55.7
$T_{1\,2}$ negative	20	11	55	5.8	9	45	18.6	56	53.2
$T_{2\,3}$ negative	24	12	50	9.7	12	50	17.5	47.0	45.0
$T_{1\,2\,3}$ negative	13	6	46.1	6.6	7	53.8	15.3	50	53.3
Grand average			49	8.27		50.9	16.1		

The results obtained in this investigation do not correspond in all details with those of Willius[9] at the Mayo Clinic. The grand average mortality given by Willius was 50.4 per cent in 11.2 months, which compares closely with 49 per cent in 8.27 months in this series. On the other hand, Willius records the highest mortality in the T_1 negative group, whereas our highest is in the $T_{1\,2}$ negative group. We found the mortality in the $T_{2\,3}$ group much higher than did Willius: in fact, even higher than in the T_1 negative group. Negative T-waves in combined Leads I and III or in Lead II alone have not been observed.

There can be no doubt as to the seriousness of the prognosis in patients showing negative T-waves in the various leads tabulated. Many of the patients examined did not present obvious signs of serious heart disease, and a prognosis based upon the clinical findings alone, unaided by electrocardiographic examination, would be fallacious.

It is to be noted that the average age of the dead and living in each group is about the same. The only feature regarding age which might appear to be significant is that in the $T_{2\,3}$ negative group the average age is definitely lower than in any of the others.

BUNDLE-BRANCH BLOCK

The electrocardiograms in this series showed widening of the QRS interval beyond 0.10 seconds in all leads. Notching of the QRS waves was usually present but electrocardiograms showing notching with-

out widening have not been included. The T-waves were usually opposite to the main QRS deflections in all leads, and there was in all cases a definite ventricular preponderance.

Thirty-seven patients have been traced. Twenty-eight of these had arteriosclerosis, 5 rheumatic heart disease, 3 syphilis, and 1 bacterial endocarditis. Seventy-three per cent are dead, the average duration of life being 5.5 months, with a maximum of 23 months. Twenty-seven per cent are known to be alive, the average duration of life being 14 months, with a maximum of 27 months. These figures are in general accord with previous observers.[3, 4, 8] In this condition, also, the clinical diagnosis unaided by electrocardiographic examination would be most uncertain, if not impossible. A considerable number of these patients, however, did present a poor first sound, which was frequently split, at the mitral area, also an apical systolic murmur. These findings in a patient over forty-five years of age, who also gives a history of progressive symptoms of myocardial failure extending over a period of months, and in whom no other predominating etiological factor is discoverable, are suggestive of bundle-branch block.

The mortality recorded in this group indicates the gravity of the condition, and patients whose electrocardiograms show existing defects of the conducting mechanism within the ventricle are to be given a most guarded prognosis.

AURICULAR FIBRILLATION WITH EXTRASYSTOLES

In a paper by George W. Norris,[10] a statement is made that the mortality in patients with fibrillation accompanied by ventricular extrasystoles is as high as the mortality in pulsus alternans, setting the latter figure at 100 per cent within 3 years. All patients having auricular fibrillation with extrasystoles, in whom complete statistics regarding duration of life could be obtained, have been studied. A larger group of patients with auricular fibrillation uncomplicated by extrasystoles has been tabulated for comparison. Patients receiving digitalis within two weeks prior to examination have been excluded. Of the 20 patients traced, 15 had arteriosclerosis, 4 rheumatic heart disease, and 1 syphilis. Sixty per cent of these patients died, having an average length of life, after the first examination, of 13.3 months, the maximum being 37 months. Forty per cent are known to be alive, an average time of 23.4 months and a maximum of 54 months having elapsed since first coming under observation. For comparison a group of patients with fibrillation of the auricles without extrasystoles has been studied. Of these 53.8 per cent died within an average time of 14.4 months after having first come under observation, the maximum duration of life being 58 months. Those still living comprise 46.2 per cent of the total, the average length of life being 38.3 months, and the maximum 60 months.

Comparing the two groups of auricular fibrillation studied, the percentage mortality in those patients having fibrillation with extrasystoles is certainly higher than is the case where extrasystoles are not present. If, however, we take into consideration the fact that the average age of the former group is 60.5 years as compared with 46.5 years in the latter, it would appear evident that the higher mortality might very definitely be associated with the increase in age and not with the presence of extrasystoles.

LOW VOLTAGE

The observation that, not infrequently, the electrocardiograms of patients with clinical signs of serious heart disease show small amplitude of the main ventricular wave* led to the study of low voltage cases. There was no recognized criterion upon which to base an electrocardiographic diagnosis of low voltage and for this study a maximum excursion of 5 mm. in the ventricular wave has been adopted as the standard. The usual standardization of 3 cm. deflection for 3 mv. of current was used in all cases. From this group are excluded: cases of bundle-branch block; negative T-waves, except in Lead III, and cases of auricular fibrillation with extrasystoles. Twenty-two of the 34 patients were arteriosclerotic and 8 rheumatic; of the remainder, 1 was a case of veronal addiction, and another a case of duodenal ulcer in which the only evidence of cardiac involvement was a history of paroxysmal tachycardia. Both patients are alive, the veronal case having been under observation for 56 months. Of these patients 67.6 per cent died cardiac deaths in an average of 6.17 months, the length of life varying from less than one month to 34 months; 32.4 per cent are alive, the average length of life being 23.2 months, with a maximum of 56 months.

One of the patients having bundle-branch block and not included in this low voltage group, has shown very interesting variations in voltage. He is a man forty-seven years of age and has been under continuous observation for almost 2 years, during which time he has had 3 periods of very marked myocardial failure with extreme dyspnea and edema. His voltage has varied directly with his clinical condition, being very low when the myocardial failure was marked and rising to normal values when the signs of failure improved markedly or disappeared.

A study of the mortality and the average duration of life leaves no doubt as to the gravity of low voltage as a prognostic sign. In fact, the only group with a higher mortality is that of bundle-branch block.

*Since this work was completed an article by Master and Pardee, mentioning the significance of low voltage has appeared in Arch. Int. Med., 1926, xxxvii, 42.

TABLE II

	Cases heard from	PATIENTS DEAD			PATIENTS LIVING				
		No. of cases	Percentage	Average length of life in months	No. of cases	Percentage	Average length of life in months	Average age of dead in years	Average age of living in years
Bundle-branch block	37	27	73	5.5	10	27	14	57	57.4
Fibrillation with extrasystoles	20	12	60	13.3	8	40	23.4	60.5	55.6
Fibrillation without extrasystoles	39	21	53.8	14.4	18	46.2	38.3	46.5	53.8
Low voltage	34	23	67.6	6.17	11	32.4	23.2	56.7	55.2

CONCLUSIONS

1. Negative T-waves in various combination of Leads I, II and III, other than in Lead III alone, warrant a grave prognosis. The mortality figures in the present study are comparable to those of other observers.

2. Signs of bundle-branch block would appear to be the most serious electrocardiographic abnormality.

3. The presence of ventricular extrasystoles does not appear to increase the mortality in auricular fibrillation.

4. Low voltage unaccompanied by other electrocardiographic abnormalities is a prognostic sign of serious import.

We wish to express our indebtedness to Dr John Oille for his assistance and for the permission to include in the records given a number of his private patients: also to Professor Duncan Graham for many helpful suggestions.

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A COMPARATIVE STUDY OF "RHEUMATIC" MITRAL REGURGITATION AND MITRAL STENOSIS* †

HOWARD B. SPRAGUE, M.D., AND PAUL D. WHITE, M.D.
BOSTON, MASS.

TO PRESENT a study of mitral regurgitation and stenosis is to risk reopening a perennial discussion. Fashions in medical diagnosis are so variable and beliefs of different men so actively defended that it is difficult to take a reasonable middle course. Most surely is this true in the diagnosis of organic mitral regurgitation.

In former days the diagnosis of mitral regurgitation was more commonly made than any other in heart affections. Any apical systolic murmur was sufficient to justify the observer in attaching this label and with it the dire prognosis attending a "leaky valve." The indiscriminate use of this diagnosis, without doubt, was responsible for a great deal of harm.

With the coming of the World War it became evident to recruiting officers, after the loss of much valuable time, that the widespread finding of mitral regurgitation in examinations for the various branches of the service and the resulting rejection of many men was limiting the available recruits to a dangerous extent. Then the tide began to turn in the other direction and, following the lead of Sir Thomas Lewis[1] and Sir James Mackenzie,[2] it became the vogue entirely to disregard systolic murmurs at the apex of the heart in otherwise healthy young persons. So far did this opinion spread that apical systolic murmurs were often not even noted on physical examinations and loud bruits were dismissed in the broad phrase "no significant murmurs heard." Indeed students trained during the later years of the war, and even to the present, were taught not to make the diagnosis of organic mitral regurgitation but to say, whenever the mitral valve was diseased, no matter what the clinical findings were, "This is a case of mitral stenosis."

Of late years this viewpoint has been taken by many, among others by Dr. Richard Cabot,[3] whose sweeping statements about the nonexistence of uncomplicated regurgitation have gone far to throw this diagnosis into the discard. However, he supports his belief that the condition is rarely, if ever, a clinical entity by autopsy statistics, which are shown to be of doubtful value by his further agreement with Mackenzie that "no one ever dies of mitral regurgitation." One might almost as well say that there is no such thing as effort syndrome because necropsies are not reported on such cases. Furthermore it should be said that the autopsy records of the Massachu-

*From the Cardiac Clinic of the Massachusetts General Hospital.
†Read before the American Heart Association, Dallas, Texas, April 20, 1926.

setts General Hospital do not accurately measure the incidence of mitral regurgitation, since the Children's Medical Service is relatively small, and it is just at this age period of five to twelve years that organic mitral regurgitation is most common. In fact, Poynton and Payne[4] in a necropsy series of 150 cases of rheumatic heart disease under the age of twelve found that "a marked degree of mitral stenosis" was rare, and in 11 cases "marked mitral regurgitation" was noted. Bass[5] believes that one-third of the cases of mitral insufficiency in children are unaccompanied by stenosis.

Dogmatic pronouncements, such as those of Lewis and Cabot, are of value in national emergencies and in civilian life in diverting medical attention away from unimportant physical findings, yet they are not without danger. They appeal to the medical students and to those practitioners who are only too willing to accept short-cuts in diagnosis and are averse to the careful weighing of evidence. It was with the desire to determine the wisdom of establishing an accurate clinical entity of mitral regurgitation as distinct from mitral stenosis that our investigation was undertaken, chiefly because of our enlightening experience during the past few years in the Children's Cardiac Clinic at the Massachusetts General Hospital.

We have felt that mitral systolic murmurs cannot always be lightly dismissed. Even in such mass statistics as are furnished by life insurance examinations,[6] it can be shown that there is a definitely increased mortality rate in those people who show apical systolic murmurs, largely we believe, because definite apical systolic murmurs, even though they are not due in many cases to organic valve changes, are in most instances evidence of some sort of ill-health sufficient to interfere with cardiac tone. There is, of course, the possibility that coexisting diastolic murmurs were missed in some of these cases.

But it is with mitral insufficiency based upon actual changes in valve leaflets arising from endocarditis of the "rheumatic" type that we are mainly concerned. It should be evident that this diagnosis is an important one to make, chiefly for the reason that mitral regurgitation, in the great majority of cases, leads to mitral stenosis, and the early recognition of mitral endocarditis may stimulate efforts to limit the recurrences of rheumatic infection to which such patients are unfortunately so liable. Much less frequently the lesion remains stationary, and we find what we have called "pure mitral regurgitation" lasting beyond middle life.

DIAGNOSTIC CRITERIA

This brings us to the criteria used in diagnosis, and it is essential to recognize that we are not merely quibbling about definitions when we use the terms "pure mitral regurgitation" and "pure mitral

stenosis." We are using these terms in a strictly clinical sense, and clinical and pathological findings need not necessarily either agree or conflict, if each exists as an entity. We grant at the outset that it is difficult to conceive pathologically of mitral stenosis without some regurgitation and of organic mitral regurgitation without some stenosis (although approximation of the latter can be more readily demonstrated). A mitral valve with an effective circumference of 7½ cm. in the adult is pathologically small but may not be decreased enough either to cause a diastolic murmur or to interfere seriously with the forward current of blood. Resting upon the findings in physical examination alone, one can without doubt, in many cases, clearly differentiate the "pure" types of regurgitation and stenosis.

By these "pure" types we mean cases in which the murmurs are essentially and obviously of importance. The conclusions are based upon a very carefully selected series of 20 cases each of regurgitation and stenosis seen in clinic or consultation practice in the past year and a half. Several cases have been followed for many years and have been seen repeatedly. The findings are constant and the murmurs definite. All are ambulatory patients with normal rhythm, without obvious congestive failure or other cause for frank dilatation (with its confusing signs attending heart failure), and are all without active infection. All cases have been studied by x-ray and all but two by electrocardiogram.

We have considered a diagnosis of "pure" organic mitral regurgitation justified when there has been a constant, loud, apical systolic murmur, with enlargement of the heart recognized by physical examination or roentgenogram, with or without a frank rheumatic history, in the absence of hypertension, arteriosclerosis,* fever, anemia or other such factors that might produce dilatation. One case has been included in which a loud apical systolic murmur has persisted after an attack of rheumatic fever occurring less than a year previously, but in whom, so far, no enlargement or abnormality in the shape of the heart is demonstrable. In none of these cases is a mitral diastolic murmur audible, even after exercise, although a third sound can be heard in most of them.†

Likewise, we have said that there was "pure" mitral stenosis in those cases in which there was a definite mitral diastolic murmur (associated usually with a presystolic accentuation), but no apical systolic murmur.‡ These cases are common, particularly in adult life. Coombs[7] found no regurgitant bruit in 46.7 per cent of a series of cases with definite evidence of mitral obstruction. They were described by

*In some old people, however, mitral regurgitation may result from sclerotic involvement of the mitral valve itself.

†In two cases at a single examination a questionable inconstant mid-diastolic rumble was noted.

‡In three cases a very slight systolic murmur was at times audible.

Duroziez[8] in 1877, and "pure mitral stenosis" is known as Duroziez's disease. Enlargement of the heart or rheumatic history is, of course, less essential in this diagnosis than in that of regurgitation, and, as a matter of fact, our attention was originally directed to this problem by the observation that in many cases the apex impulse is further to the left in cases of mitral regurgitation than in mitral stenosis.

The two series were studied in parallel in regard to history, activity, symptoms, duration of heart disease, physical examination, x-ray and electrocardiogram.

For comparison of the relative incidence of these valve lesions in children it is of interest to note the approximate figures from the Children's Cardiac and Chorea Clinics of the Massachusetts General Hospital in 1925.

	NO. OF CASES
Mitral stenosis and regurgitation* combined	143
Mitral regurgitation alone (organic)†	29
Mitral stenosis alone‡	1
Mitral stenosis and regurgitation and aortic regurgitation	26
Mitral stenosis and aortic regurgitation	2
Aortic stenosis, aortic regurgitation, mitral regurgitation	1
Congenital heart disease	11
Potential heart disease	66
Questionable heart disease	34
Normal heart	53
Total	366

*Showing both mitral systolic and diastolic murmurs.
†Showing only the "organic" mitral systolic murmur (already described).
‡Showing only the mitral diastolic murmur.

AGE AND SEX

(*a*) *Mitral Regurgitation.*—The ages of these patients when they were first included in the series were as follows:

Fourteen cases were fifteen years old or younger, the youngest being six years old. Six cases were nineteen years old or older, the oldest being sixty years old. That is, almost three-fourths of this group were children.

Nine were males and eleven were females.

(*b*) *Mitral Stenosis.*—As might be expected, the patients with mitral stenosis tended to be older. Time was necessary to produce such marked narrowing of the mitral valve.

One patient was eleven and another fifteen years old. All the rest were nineteen years of age or older, the oldest being fifty. One-half of the series fell in the age group twenty-five to thirty-five.

The figures for sex are consistent with the general knowledge that mitral stenosis is commoner in females, since in our series there were only 5 males to 15 females.

HISTORY OF "RHEUMATIC" INFECTIONS

The important infections which have been searched for in the past history of these patients are rheumatic fever, chorea, tonsillitis and scarlet fever. A history of indefinite joint pains has been considered significant in patients with loud murmurs, especially if they were accompanied by fever or by a story of heart trouble having been discovered in the course of some such infection. Past histories are notoriously inaccurate, but careful questioning has been employed to gain as nearly exact a story as possible.

(*a*) *Mitral Regurgitation.*—

SINGLE AND COMBINED ETIOLOGIC DISEASES

	RHEUMATIC FEVER	CHOREA	TONSILLITIS	SCARLET FEVER
No. of cases	10	1	6	4

Tonsillitis alone was found in 3 cases, scarlet fever alone in 2 cases. One child of ten years had had many sore throats, with scarlet fever and diphtheria eight years before she was seen in the clinic, but had not been found to have a valvular lesion until after erysipelas five years later. A man of twenty years related his heart disease to diphtheria six years before, a very questionable etiology. A child of six years had had whooping cough followed by pains in the ankles three years before, but no heart disease was found by her family physician until after scarlet fever and pneumonia two years later. In one case "fever and heart trouble" had followed measles eight years before, and the patient had been in a heart hospital for eight months.

(*b*) *Mitral Stenosis.*—

SINGLE AND COMBINED ETIOLOGIC DISEASES

	RHEUMATIC FEVER	CHOREA	TONSILLITIS	SCARLET FEVER
No. of cases	9	3	9	2

In 3 cases only vague joint pains or "growing pains" were noted. One woman of fifty years was told that her heart was damaged at the age of six years, when she had diphtheria, again a questionable connection. Another had rheumatic fever and endocarditis following scarlet fever eleven years before. In one case the only history was of questionable diphtheria, and in 2 cases no history of infections could be elicited.

PHYSICAL ACTIVITY AND SYMPTOMS

The effect of the cardiac lesion upon the daily life of the patients was studied by considering their physical activity with or without symptoms. All cases were ambulatory, but most of them had some symptoms which had directed their attention to the heart.

(*a*) *Mitral Regurgitation.*—Twelve patients in this group were active school children or persons doing work not calling for severe muscular strain, and were symptom-free. All of the remaining eight were at times troubled by some complaint referable to the heart, mainly, however, of the effort syndrome type, as follows:

	CASES
Slightly more dyspnea on exertion than normal	4
Palpitation	2
Easy fatigue	2
Irregularity of the heart	1
Faintness and dizziness	1
Paroxysmal fibrillation	1
Pyrosis	1
Epistaxis	1

(*b*) *Mitral Stenosis.*—It is obvious that the cases of mitral stenosis have more symptoms of cardiac disability than those with pure mitral regurgitation. Only two patients were said to be symptomless, one a boy of fifteen years, the other a girl of fourteen. The main complaints of the others were:

	CASES
Dyspnea on moderate exertion	9
Palpitation	7
Paroxysmal tachycardia	5
Precordial aching, pain or pressure	4
Hemoptysis	4
Paroxysmal auricular fibrillation	1
Paroxysmal auricular flutter	1
Orthopnea	2
Irritable heart symptoms of many types	2
Easy fatigue	1
Cough	1
Recurrent laryngeal nerve paralysis	1

DURATION OF HEART DISEASE

It is, of course, impossible to be sure of the duration of cardiac infection in any group of patients who have not had repeated physical examinations during their early life. The duration of the symptoms of which the patient complains does not measure the duration of that patient's heart disease, and presumptive evidence must be taken in relation to significant infections.

(*a*) *Mitral Regurgitation.*—Of the cases in which the duration was known:

Three	had	had	heart	disease	less	than	1 year
One	"	"	"	"	for	1	year
One	"	"	"	"	"	3	years
One	"	"	"	"	"	6	"
Two	"	"	"	"	"	8	"
One	"	"	"	"	"	17	"
One	"	"	"	"	"	30	"

The probable duration of heart trouble in 9 other cases indicated that:

Four	cases	had	had	it	for	at	least	3	years
Three	"	"	"	"	"	"	"	4	"
One	case	"	"	"	"	"	"	5	"
One	"	"	"	"	"	"	"	20	"

In one patient the duration was entirely unknown. It will be seen that three-fourths of this group had probably had their heart lesions for over three years, and three of them for over seventeen years.

(*b*) *Mitral Stenosis.*—It was not possible to make a reasonable guess as to the duration of the lesion in 6 cases of stenosis. The minimum durations of 5 that were known were the following:

One case	8	years
Two cases	11	"
" "	12	"

The hearts of 9 others had probably been damaged at least as long as these figures indicate:

One case	3½	years
" "	4	"
" "	19	"
Two cases	20	"
One case	24	"
" "	29	"
" "	30	"
" "	44	"

All but two cases in whom the duration was known or reasonably suspected had had heart disease for over eight years, and five for over twenty years, a longer duration than in the case of mitral regurgitation.

It is of interest to note that one girl of twenty, who was seen for twelve years in the clinic, showed in 1912, after mild chorea, a blowing apical systolic murmur. Six years later a questionable mitral diastolic murmur could also be heard, indicating stenosis; and four years after that the systolic murmur was gone, leaving only the rumbling diastolic and presystolic murmurs of pure mitral stenosis.

PHYSICAL FINDINGS

Consideration of the physical examination of the heart in these cases brings up the question of the physiology involved in the production of heart murmurs. Murmurs are set up whenever a stream of fluid passes from a tube of a given caliber into one of a markedly different diameter. The origin of both systolic and diastolic murmurs is therefore essentially the same. The intensity of the murmurs at the valve is largely dependent upon (*a*) the size of the opening,

(*b*) the velocity of the fluid current, which in turn is related to the difference in pressures on either side of the narrow part of the channel, and (*c*) the size of the chamber beyond the obstruction. The underlying processes involved in the production of murmurs at any valve are well brought out by Thayer[9] in relation to the normal systolic murmurs heard at the base of the heart, caused by the discharge of blood through a relatively small opening into a distensible vessel above. Blowing systolic murmurs at the mitral area are doubtless, in most cases, caused by regurgitation through the mitral valve. In inflammatory changes of the valve cusps or shortening of the chordae tendineae it is easy to see that the valve cannot close accurately and will allow blood to pass back into the auricle during ventricular systole. During acute infections or in failing hearts another mechanism is in action, namely, the dilatation of the chambers and the stretching of the valve ring. The leaflets do not accurately oppose during systole and leakage occurs. For this reason it can be said roughly that the smaller the heart in which a loud apical systolic murmur occurs the more significant the murmur is of actual valve damage. It is probably true that in many normal hearts there are congenital abnormalities of the cusps of a minimal amount which interfere with perfect coaptation and permit a small amount of regurgitation. This would account for many so-called functional systolic murmurs in normal young people, and it is suggested that the audibility of these murmurs is mainly dependent upon youthful tachycardia and thinness of chest walls. Apical systolic murmurs originating outside the heart, or not transmitted from the base of the heart, we believe to be relatively rare and the mechanics of their production obscure, although a few are probably due to displacement of air in the lung as the result of the heart beat.

Diastolic murmurs of mitral origin are indicative of some narrowing at the valve orifice, but like the systolic murmur (though in rarer cases) may at times be due to dilatation of the heart chambers with a relative stenosis of the valve. Wood and White[10] have published descriptions of four such cases in which a mitral diastolic murmur was heard in very much enlarged hearts.

Mitral diastolic murmurs are low-pitched and rumbling and for that reason are easier to miss on auscultation than are the higher-pitched systolic murmurs. Although other factors, such as the condition of the valve leaflets usually found in mitral stenosis, are important, it is nevertheless true that the lower pitch of the diastolic murmur is largely due to the relatively slower blood current passing through the valve during diastole as contrasted with the high pressure jet through an incompetent valve in ventricular systole. It is

of interest in this connection to remember that the murmur of mitral regurgitation is essentially a murmur of stenosis, since the blood is forced back through a narrow opening into a large chamber (the auricle) beyond.

For the production of a loud murmur in either cardiac phase there must exist an optimum relationship between auricle, ventricle and valve, as well as an active blood flow. It is conceivable that such stretching of the mitral ring can exist in cardiac dilatation, coincidently with a slowing of the blood current, that no systolic murmur will be audible. This helps to account for the absence of a murmur in cases in which necropsy shows obvious opportunity for regurgitation.* It is again not infrequent to find necropsy cases in which marked mitral stenosis is present and in which no diastolic murmur was described. In most of those cases we feel that a faint mid-diastolic murmur was missed on examination, or that the congestive failure was so marked, especially with auricular fibrillation, that the blood flow was insufficient to cause the vibrations at the stenotic orifice.

These considerations have some bearing on the explanation of the absence of systolic murmurs in clinically "pure" mitral stenosis. Advanced mitral stenosis leads invariably to left auricular dilatation and stasis. It is probable that the diastolic remainder in the auricle in some way interferes with the production of a murmur by the small regurgitant stream which may pass back. Onflow from the distended auricle into the completely empty ventricle, however, results in a murmur. In rare cases the valve ring may be stenotic, but the cusps sufficiently flexible to prevent regurgitation of any extent.

The systolic murmur occurring in the cases of our series showing mitral regurgitation was in all instances a loud, blowing, or musical murmur. It usually came directly after the first sound but in five cases it completely masked the first sound. In one patient the murmur was loudest in late systole.

The diastolic murmurs in stenosis were also loud, and were usually of the type described as "presystolic rolls," but if the heart is slow one can always hear such murmurs begin in mid-diastole.

The third sound is such a common finding in young people that it is not considered abnormal. It occurred in almost all cases of both regurgitation and stenosis. It is exaggerated in mitral stenosis, and a loud third sound in a patient with a definite apical systolic murmur is suggestive, but by no means diagnostic, of valvular deformity.

*As Thayer[8] has pointed out, the propagation of murmurs in the direction of the blood current also makes mitral systolic murmurs less easily audible at the apex, because of their transmission to the back.

Enlargement of the heart is most easily determined clinically by measurement of the displacement of the left border, as indicated by the position of the apex beat. We have considered the midclavicular line as the boundary of the normal left border. This comparison coincides so well with the cardiothoracic ratio found by roentgenogram that it will be discussed under the section on x-ray. It is common, however, to find the apex beat outside the midclavicular line in mitral regurgitation of more than a few months standing.* On the other hand mitral stenosis of long duration may not result in displacement of the left border.

In no case of either series was hypertension a factor as far as could be determined by history or blood pressure measurements.

ROENTGENOLOGICAL FINDINGS

The x-ray studies of these cases with mitral abnormalities have revealed interesting effects of the valve lesions on the shape and size of the heart. A considerable amount of work has been done by roentgenologists in this field, so that a picture of the "mitral heart" is well known. The differential effects of regurgitation and stenosis are, perhaps, less familiar.

As might be suspected, the first demonstrable effect of either lesion upon the heart is brought to bear upon the left auricle, and prominence of the shadow in this region can be seen in all but the very earliest cases. It is somewhat complicated by enlargement also of the structures involved in the pulmonary circulation. Increased pulmonary pressure leads to dilatation of the pulmonary artery and hypertrophy of right ventricle, especially in the region of the conus.

The typical changes of stenosis or regurgitation are to be seen, however, in the more advanced stages of either lesion. In mitral stenosis the left auricle becomes enlarged by hypertrophy and dilatation, and the stasis in the lungs leads to further enlargement of the right ventricle. In well marked cases the wall may become as thick as that of the left ventricle, a relationship further aided by the failure to hypertrophy, or by even the slight atrophy, of the latter chamber. Furthermore the enlarged right ventricle displaces the right auricle laterally and as it dilates it leads to auricular dilatation by incomplete emptying or tricuspid regurgitation. In advanced cases the right ventricle, especially at the conus, may show as a bulging prominence on the left ventricular border by roentgenogram.

*We have included one borderline case in which the lesion is of only 6 months duration and in which no enlargement is as yet demonstrable by physical examination or x-ray.

A different picture is typical of mitral regurgitation and is marked by the changes in the left ventricle. The dynamics involved are obvious. The ventricle, to keep up an effective circulation, is forced to throw out with each beat a larger amount of blood than it does under normal circumstances, because of the loss of blood into the left auricle. As far as the effect upon the ventricle is concerned, the work involved results in the same compensatory change as occurs in aortic regurgitation, and as a matter of fact the auricular enlargement, as seen by x-ray, is overbalanced by the well-marked left ventricular hypertrophy. It is probably fair to say that in any case of uncomplicated mitral disease which shows this left ventricular enlargement regurgitation is, or has been, a significant factor. Although no proof can be at present produced, it is suggested that in those cases of "pure" mitral stenosis in which the left ventricle is small, the stenotic process has been of relatively rapid development and the regurgitation has not persisted long enough to produce left ventricular hypertrophy. Further support is given to this hypothesis by the fact that the large majority of cases of mitral disease which, as we have seen, show both systolic and diastolic murmurs have also both ventricular and auricular enlargement by x-ray.

A further complicating factor is possibly of importance in these x-ray determinations. In advanced mitral stenosis the right ventricle may hypertrophy so markedly that it is enlarged downward and to the left, as well as to the right, so that it may cause a confusion with the left ventricle along the left side of the heart shadow, or may by its enlargement displace the left ventricle laterally and thus increase the total transverse diameter.

Comparison of the average of the ordinary measurements of the heart x-rays of the two groups does not show very significant differences, and it is only by reference to the plates themselves that the characteristic changes become evident. Review of these roentgenograms by Dr. G. W. Holmes, roentgenologist at the Massachusetts General Hospital, showed that in at least one-half of the cases of mitral regurgitation a definite prominence of the left ventricular region was to be seen. In only about one-fourth of the cases of "pure" stenosis was this suggested. Changes in the region of the left auricle and pulmonary artery were more striking in the latter group. Enlargement to the right of the midline is found in both series, proportionately slightly more in those with regurgitation.

The average figures are as follows:

	M.R.	M.L.	T.T.	L.	B.	GV.	CHEST
Regurgitation	4.0	7.5	11.5	12.9	9.7	5.4	20.6
Stenosis	4.1	8.5	12.6	14.2	10.7	5.7	23.6

If we use the cardiothoracic ratio, assuming that the total transverse diameter should not exceed one-half the internal diameter of the chest, we find that in the cases of regurgitation the average heart shadow is 1.2 cm. too wide, while in stenosis it is only 0.8 cm. too wide. There seems in this series, therefore, to be a slight tendency for the heart to be larger in transverse diameter in relation to the chest in the former than in the latter condition.

These findings are by no means a new discovery, but they suggest the importance of making x-ray studies in young patients with loud apical systolic murmurs. The early changes in heart size, particularly in the region of the upper left border, are valuable evidence of the organic nature of the murmur, while enlargement of the left ventricle may be found as the lesion progresses.

ELECTROCARDIOGRAMS

(*a*) *Regurgitation.*—All cases studied showed normal rhythm by electrocardiogram. In three, due to temporary nervousness, a sino-auricular tachycardia with a rate of 130 was noted. Sinus arrhythmia was marked in three others.

The axis deviation (by method of "index" or angle) is shown in the following table:

	CASES	INDEX	ANGLE	TOTAL
Slight right axis deviation	1	−12	+75°	1
	1	−11	+90°	
	1	−12	+90°	
	1	−11	+90°	3
Left axis deviation	1	+39	− 4°	
	1	+19	−53°	
	1	+24	+18°	3
No axis deviation	13			13
				20

In no case was right axis deviation marked, and in two cases the tendency to left sided preponderance was obviously abnormal.

Other abnormalities noted were:

High origin of T_2	2
Low T_2	2
High T_2	3
P_1 high and notched	1
T_2 doubled	1
P-R interval 0.22 sec.	1

(*b*) *Stenosis.*—In the series of cases with stenosis one had ventricular premature beats recorded and one other had marked sinus arrhythmia, with a ventricular premature beat and retrograde auricular contraction.

Axis deviation was as follows:

	CASES	INDEX	ANGLE	TOTAL
Right axis deviation	1	−16	+103°	
	1	−15	+130°	
	1	−15	+ 90°	
	1	−23	+123°	4
Slight right axis deviation	1	−11	normal	
	1	− 8	normal	
	1	−11	normal	3
Slight left axis deviation	1	+23	normal	1
No axis deviation	10			10
				18

The greater preponderance of right sided activity is indicated by these figures. Well marked right axis deviation appears in four cases. In only one case is left axis deviation noted and then only by index, not by angle.

Other abnormal findings:

Prominent P waves in Leads I or II	6
Low T_2 (digitalis)	3
High T_2	1
Partial A-V block (digitalis)	2

These electrocardiographic observations may be taken as suggestive evidence in corroboration of the findings that mitral regurgitation has a significant effect upon the left ventricle as well as the right heart and that mitral stenosis produces its most marked changes in structures comprising the base and right ventricular region.

SUMMARY AND CONCLUSIONS

1. It is often possible to differentiate, clinically if not pathologically, cases of "pure" mitral regurgitation, and "pure" mitral stenosis, by means of the murmurs heard on auscultation.

2. Loud apical systolic murmurs unaccompanied by diastolic murmurs cannot be entirely disregarded, as has often been done, but should be judged in relation to history, other physical findings, and x-ray measurements of the heart.

3. Twenty selected cases each of "pure" mitral regurgitation and "pure" mitral stenosis have been studied by history, physical examination, x-ray, and electrocardiogram. A review of the findings is given.

4. Mitral regurgitation causes a general enlargement of the heart, as contrasted with the right ventricular and left auricular enlargement due to mitral stenosis.

5. Pure organic mitral regurgitation is a clinical entity but is not demonstrated at necropsy as it is rarely if ever fatal. This does not,

however, decrease its significance, since regurgitation is usually but one stage in the process of stenosis which is more often the terminal condition. Under the most favorable conditions organic mitral regurgitation exists as a potential danger.

We wish to express our appreciation of the cooperation of Dr. T. D. Jones and Miss Edith Terry in the collection of some of our data.

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(*For discussion see page* 649.)

FURTHER OBSERVATIONS ON PATIENTS WITH HYPERTENSION AND INCREASED BASAL METABOLIC RATE*

ERNST P. BOAS, M.D., AND SHEPARD SHAPIRO, M.D.
NEW YORK, N. Y.

LAST year we reported upon a group of patients with true hypertension associated with an elevated basal metabolic rate, tachycardia, nervousness, loss in weight and pigmentation of the skin.[1] We wish at this time to report additional cases and present further evidence to justify our conclusions that these symptoms constitute a special syndrome which should be distinguished from the ordinary form of hypertension, as well as from exophthalmic goiter. Since our publication, our attention has been drawn to a paper by Riesman,[2] which appeared in 1919, in which he described cases apparently similar to ours under the term "nongoitrous thyrotoxic hypertension." He says, "This form occurs with preponderating frequency in women. The patients are near or past the menopause, are not overfat, sometimes spare, often single. They complain chiefly of palpitation and headache; are emotional, and have a tendency to sweating. The heart is rapid, even to the point of tachycardia; the hands are tremulous. There is often a von Graefe sign, but no exophthalmus and no goiter or other tangible abnormality of the thyroid gland. The systolic pressure ranges from 150 to 210, with a diastolic pressure of from 90 to 120." Riesman did not measure the heat production in any of his cases. Liljestrand[3] has recently reported increased basal metabolism and minute-volume flow in five patients with hypertension.

Hamburger[4] has found an elevation of the basal metabolic rate in about three-quarters of all patients with cardiac insufficiency and has suggested that the rise in heat production parallels the degree of cardiac failure. In order to bring further proof that the high readings in certain of our patients with hypertension were not conditioned by this factor, we studied the basal metabolic rate in 42 patients with heart disease in all stages of circulatory sufficiency and insufficiency. These are being reported in detail by one of us in another place. Suffice it to say that we never observed an elevation of metabolism in patients with heart disease, even when their cardiac reserve was poor, provided that the determination was made under actual basal conditions. Only patients with real dyspnea at rest showed increased heat production, and this is readily explained by the muscular exertion and

*From the Medical and Laboratory Divisions of Montefiore Hospital for Chronic Diseases, New York.

BASAL METABOLISM IN PATIENTS WITH HYPERTENSION

NO.	NAME	SEX	AGE	BLOOD PRESSURE		BASAL METABOLIC RATE	PULSE RATE	CARDIAC RESERVE	WEIGHT LBS.	LOSS IN WEIGHT	REMARKS
				Syst.	Diast.						
1	R. F.	F	40	170	90	− 4	80	Good	111	None	Mitral stenosis
2	S. S.	F	30	170	98	+ 4	90	Poor	120	None	Mitral stenosis
3	M. S.	F	47	190	110	+12	96	Good	180		
4	E. G.	F	48	190	110	+ 8	90-100	Good	202	None	
5	S. S.	F	52	220	120	−10	90	Good	142	40 lbs. in 2 yr.	
6	S. L.	F	53	250	120	− 2	78	Fair	125	40 lbs. in 1 yr.	
7	B. B.	F	55	230	110	− 8	84	Good	217	None	On 2/17/25
				164	106	− 4	90	Fair	185	32 lbs. in 11 mo.	On 1/12/26
8	A. F.	F	60	175	96	+ 4	70-80	Fair	115	29 lbs. in 2½ yr.	
9	N. P.	F	60	235	100	− 5	80	Good	127	19 lbs. in 5 yr.	Diabetes
10	R. S.	F	63	180	75	+10	90	Fair	190	None	
11	R. H.	F	65	210	120	− 1	80	Fair	173	None	
12	J. B.	M	51	182	88	+ 1	70	Good	199	15 lbs. in 2 yr.	
13	M. F.	M	52	190	100	− 1	80	Poor	122		
14	P. B.	M	53	178	100	− 4	80	Good	204	50 lbs.	
15	I. S.	M	54	225	130	+10	70-80	Good	114		
16	D. L.	M	61	166	80	− 2	86	Poor	204	30 lbs. in 1 yr.	On 12/21/22
				164	96	− 2	80	Poor	204		On 3/31/25
17	P. S.	M	66	260	140	+ 2	70-80	Good	122	None	
18	M. F.	M	34	210	145	+ 5	80-90	Fair	131		Chronic glomerulonephritis
19	A. T.	F	34	240	130	+15	96	Poor	98		On 9/2/25 chronic glomerulonephritis
				250	150	+17	96	Poor	98		On 9/24/25

BASAL METABOLISM IN PATIENTS WITH HYPERTENSION—CONT'D

NO.	NAME	SEX	AGE	BLOOD PRESSURE		BASAL METABOLIC RATE	PULSE RATE	CARDIAC RESERVE	WEIGHT LBS.	LOSS IN WEIGHT	REMARKS
				Syst.	Diast.						
20	M. D.	F	33	160	95	+56	124	Good	122.5	Yes	
21	J. W.	F	33	166	110	+43	96	Good	145		
22	R. H.	F	42	280	180	+45	96	Fair			On 4/2/25
				270	180	+39	96	Fair	88	31 lbs. in 6 mo.	On 4/7/25
				217	142	+34	88	Fair			On 5/26/25
23	E. I.	F	45	210	110	+86	120	Good	89	70 lbs. in 7 mo.	On 3/11/24
				240	135	+84	140	Good	117		On 3/12/25
				230	130	+49	134	Good	137		On 11/20/25
24	B. J.	F	50	220	116	+18	100	Good	110		On 6/6/22
						+38	90	Good	106.5		On 6/27/22
25	S. W.	F	54	175	100	+25	85	Fair	121	Little	On 12/20/23
				185	120	+26	108	Good	130		On 11/17/25
				180	98	+29	100	Fair	120		On 3/31/26
26	J. S.	F	56	174	92	+22	124	Good	123	75 lbs. in 10 yr.	On 2/5/24 diabetes
						+44	108	Good	125		On 3/20/24
				210	90	+38	90	Good	126		On 3/16/26
27	E. B.	F	65	204	104	+27	80	Poor	228	18 lbs. in 3 mo.	On 1/29/24
				205	105	+39	92	Fair	214		On 5/14/25
				195	100	+33	80	Fair	160		On 11/24/25
28	H. L.	M	37	190	120	+34	90	Fair	150	25 lbs. in 6 yr.	
29	M. N.	M	55	240	140	+33	96	Fair	127		On 9/15/25
				255	130	+30	96	Fair	112.5		On 11/24/25

mental distress accompanying the difficult respiration. This is in agreement with the findings of Grafe.[5] Only one patient of our series of hypertension cases showed marked respiratory difficulty while at rest. In her case the metabolic rate was plus 36 and plus 40 per cent.

Omitting this case, twenty-seven patients with essential hypertension and two with chronic glomerulonephritis and hypertension were examined under basal conditions. More than the usual precautions were taken. Each patient, in the postabsorptive state, was brought to the metabolism room in a wheel-chair. In order to allay the feeling of apprehension, anxiety, or fear, which occurs more often in individuals with heart disease than in other classes of patients, we took particular pains to train our subjects. This is of great importance because emotional states such as those mentioned will cause a rise in heat production even in noncardiac patients. Therefore each subject was permitted to witness another individual being run for metabolism before a determination was made on himself. A minimum of twenty minutes' rest was given before the test. If the patient was excitable he was run once, or, if necessary, twice before the actual basal metabolic rate was determined. We found a metabolic rate as high as plus 30 per cent in one excitable individual before she was trained, and a normal basal metabolism after the second run, as described above.

The Tissot spirometer and Haldane gas analysis apparatus were used in our work. Each result reported represents two successive checks. The respiratory quotients were between 72 and 93.

The two patients with glomerulonephritis had normal heat production. This is in agreement with the findings of Aub and Dubois,[6] but at variance with the report of Nonnenbruch,[7] who found elevated basal metabolism in eight patients with nephritis and hypertension.

Of the twenty-seven patients with essential hypertension ten exhibited basal metabolic rates above plus 15. In most cases with increased metabolism several estimations were made on different days. Two of the eight males and eight of the nineteen females of the series showed increased heat production. The distinguishing features of the five patients with high basal rates described in our previous communication were hypertension, tachycardia, loss in weight, pigmentation of the skin, and nervousness. During the past year we have encountered five additional cases and have made repeated observations on one of the original patients. Three of these new patients were well nourished, two of them were really obese, but all of them gave a history of having lost considerable weight.

We wish to report the progress of one patient whose case was fully reported last year, and to record the histories of two additional cases.

CASE 23.—E. I., aged forty-seven years, reported last year, was seen again in November, 1925. She weighed 137 pounds, a gain of 60 pounds over her admission weight in March, 1924. She was less irritable and nervous, although her pulse rate

was 144. The blood pressure was 220 mm. systolic and 120 diastolic. The left eye had become a little prominent, and there was a lagging of the upper lid. However, the patient winked repeatedly during the descent of the eyelid. There was a presystolic gallop rhythm. The lips had a cherry red color and there was slight cyanosis of the face. The liver was not palpable. There was a definite tremor of the hands. The thyroid gland was not enlarged. In March, 1924, the basal rate was plus 80 per cent and in March, 1925, it was plus 84; on November 20, 1925, it was plus 49.

CASE 29.—M. N., a man aged fifty-five years, whose family and past history were irrelevant, was admitted to Montefiore Hospital June 14, 1925, for treatment of an epulis. For the past six months he had complained of cough and occasional swelling of the ankles. During this time he slept poorly and had been worrying a great deal about family matters. During the six weeks before admission he complained of dyspnea on exertion and several attacks of precordial pain radiating to the left shoulder. He did not know whether he had lost much in weight.

The patient was a poorly nourished man with a skin of muddy brown color. There was no exophthalmos, and the thyroid was not palpable. The lungs were clear. The heart was considerably enlarged to the left, with a loud musical systolic murmur at the apex and a systolic murmur at the base. The right pulse was smaller than the left one. The pulse rate was 80. The blood pressure was 275 mm. systolic and 180 diastolic. The liver was felt three finger breadths below the costal margin. On the upper surface of the right lower gum there was a firm, hard, irregular mass extending from the lateral incisor to the third molar, which, on section, proved to be an epulis. The basal metabolism taken on September 15, 1925, was plus 33 per cent. The epulis receded under radium treatment. On November 24, 1925, the basal rate was plus 30. Soon after the patient began complaining of weakness and dyspnea and gradually developed signs of progressive cardiac insufficiency and died on February 13, 1926.

CASE 26.—J. S., a woman fifty-six years of age, was admitted to Montefiore Hospital on February 1, 1924. Her mother had had diabetes and one sister has diabetes. Another sister died of kidney trouble. The patient had had a goiter for thirty years, ever since the birth of her first child. Sixteen years ago she suffered a "stroke" which left her with a left-sided paralysis, which gradually diminished in degree. Six years before her admission she developed diabetes.

Examination revealed a well developed woman weighing 125 pounds. There was no pigmentation of the skin and no exophthalmos. In the region of the thyroid isthmus there was a globular mass the size of an egg, moving on deglutition, which was apparently a thyroid adenoma. The lungs were clear; the heart of normal size with a rate of 120. A systolic murmur was audible over the entire precordium but was loudest at the apex. A systolic murmur of different quality was heard at the aortic area. The blood pressure was 174 mm. systolic and 92 diastolic. The liver was enlarged, and there was no edema of the extremities. There were signs of a residual left hemiplegia, but function of the limbs was fair. The diabetes was of only moderate severity and was controlled by a general diabetic diet.

For the first month after her admission the patient ran a temperature between 100° and 101°, due to a cystitis and pyelitis. The basal metabolic rate on admission was plus 22 per cent; on March 20, 1924, when she had been afebrile for two weeks, it was plus 44.

The patient was readmitted in March, 1926, at which time her general condition was good. There was no change in the physical signs, except that the blood pressure had risen to 260 mm. systolic and 110 diastolic and that there was a coarse tremor of the fingers. Subsequent blood pressure readings were 210/90. The basal rate on March 16, 1926, was plus 38 per cent.

This group of patients, of which the above-cited cases may serve as examples, do not present the classical signs of Graves' Disease. They lack exophthalmos, asthenia, and the fine tremor of the fingers. There is no noteworthy thyroid enlargement and no bruit or pulsation over the gland. Their characteristic feature is diastolic hypertension, and many of them suffer chiefly from the cardiovascular sequelae of high blood pressure. They differ from patients with exophthalmic goiter in certain other respects as well. In three patients of our series (Cases 20, 21 and 28) thyroidectomies were performed without relief of symptoms. In two instances subtotal extirpation was performed, and in one case (28) three partial thyroidectomies were performed in four years. Case 22 received 0.3 gm. of potassium iodide daily from May 17 to May 25, 1925, but experienced no significant drop in metabolism. White cell counts in seven of the patients did not reveal the leucopenia or lymphocytosis so often found in patients with exophthalmic goiter.

We were able to test the sugar tolerance in three instances. In one (Case 23) there was alimentary glycosuria after the oral administration of 100 grams of glucose; in another (Case 25) a slightly prolonged hyperglycemia curve; and in the third (Case 27) the sugar tolerance was normal.

All of the facts presented seem to justify the conclusion that patients with diastolic hypertension and augmented heat production present a special syndrome which should be distinguished from Graves' Disease, as well as from uncomplicated hypertension.

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Society Transactions

AMERICAN HEART ASSOCIATION
1926

THE second annual scientific session of the American Heart Association was held at Stonleigh Court, Dallas, Texas, April 20, 1926. The session was called to order at 2 P.M., by Geo. L. Carlisle, Dallas, Texas, in the absence of the President, Dr. Joseph Sailer.

The following program was presented:

DR. HUGH MCCULLOCH, St. Louis: **Gain in Weight as a Guide in the Convalescent Care of Cardiac Children.** (See page 569 for original article.)

DR. CHARLES S. STULIK, Chicago: **Collaborative Health Survey of School Children.**

DR. R. H. OPPENHEIMER, Atlanta, Georgia: **Public Health Aspects of Heart Disease.**

DR. EMMET F. HORINE, Louisville, Kentucky: **The Prognosis of Heart Disease.** (See page 617 for original article.)

DR. T. HOMER COFFEN, Portland, Oregon: **Early Signs and Symptoms of Heart Failure.**

DR. HOWARD B. SPRAGUE, Boston: **A Comparative Study of "Rheumatic" Mitral Regurgitation and Mitral Stenosis.** (See page 629 for original article.)

DR. LOUIS E. VIKO, Salt Lake City: **Cardiac Neurosis Associated with "Organic" Heart Disease.** (See page 539 for original article.)

DR. J. J. SAMPSON, DR. R. L. MCCALLA, DR. WM. J. KERR, San Francisco: **Phonocardiograms of Fetal Hearts.**

DR. GEORGE R. HERRMANN: **The Austin Flint Phenomena—Experimental and Clinical Data of Its Mechanism.**

DISCUSSION

DR. JOHN H. MUSSER, NEW ORLEANS.—The first paper I heard was that of Dr. Horine, and I was very much pleased to hear him say that in aortic syphilitic disease he found the prognosis extremely grave. I think we are all coming to recog-

nize that fact. I think, however, that his explanation of why it is so grave was not entirely complete, because he did not mention the fact that the spirochetes are usually found very thickly throughout the myocardium itself, as well as in the coronary arteries.

It might be interesting to bring out the fact that we believe we have had considerable success recently in our syphilitic heart cases by treating them with bismuth, and we have had excellent results in the acute congestive cases with ouabain. I do not think we have added materially to the life of the individuals, but we have carried them along perhaps longer than otherwise.

Dr. Horine brought out the fact that if we are going to be successful in combating heart disease, periodic health examinations are necessary; and the paper following brought out the fact that health examinations will not obtain so much in the way of symptoms of heart enlargement, etc., as they will bring out the occurrence of symptoms which really are more indicative of early heart failure—that it is the symptoms rather than the signs which are of importance. If this be true, and if health examinations are going to be used extensively throughout the country and by a large body of men who have not been trained in cardiological diagnosis, then I think it would be well for the members of this Association to accentuate the point that a great deal of information may be secured from the symptoms.

Dr. Coffen dealt almost entirely with the symptoms, and with the very important one of dyspnea. I remember years ago when I was a medical student, Mackenzie's book, in which he laid great stress on dyspnea as an important early sign of beginning heart failure, had just come out. Mackenzie goes so far as to say in his former work that it is dyspnea rather than any objective sign that we can develop, which will point to the heart and to heart failure. I think that point should be stressed among the general medical public.

I am particularly interested also in the frequency with which we find gastrointestinal symptoms. At Baylor today Dr. Billings spoke, discussing medical education. He said medical education occurred in definite cycles, and the cycle which he was responsible for instituting about twenty years ago was a cycle which was bound to develop; we have now reached the acme of that cycle. New developments will occur every ten or fifteen years. He also stressed the fact that the most important change we could bring about in medical education at the present time would be to spend time developing the man who has a general knowledge of medicine and who is not primarily a specialist. He referred not only to the specialties, *per se*, the otolaryngologists, the urologists, and so on, but also to the internist, stating that there is a great tendency to teach especial internal medicine, to specialize in medicine. I do not quarrel particularly with the cardiologist, but I do quarrel with the gastroenterologist, and I think this paper brings out particularly the frequency with which early gastric symptoms are associated with cardiac failure. I have had the opportunity of seeing a rather large number of cases which have been treated extensively for gastric symptoms, when there was no question whatsoever that the gastroenterologist has missed the fact that the symptoms were probably entirely secondary to the cardiac condition present.

I was also interested in the change of sentiment which is set forth in Dr. Coffen's paper in regard to the importance of ectopics. Extrasystoles have been going through a cycle also. At one time they were always indicative of serious heart disease; then the pendulum swung the other way and ectopics became of no importance whatsoever; they were disagreeable subjective symptoms in some cases, of phenomenal interest to the man who was not accustomed to hearing them, but otherwise they were discounted. And now the pendulum has swung the other way, and

Dr. Coffen has brought out that in an equal number of his cases they are indicative of myocardial conditions.

Dr. Sprague's paper was scientific and interesting. There is little I can say about it. I would like to ask him why the diastolic murmurs are not heard as frequently as the systolic. His explanation was that these low-pitched diastolic murmurs were easier to hear than the high-pitched. My conception of the physics of acoustics is that the low-pitched note is usually easier to hear than the high-pitched. If a fife and drum corps comes down the street you hear the drum before the fifes, and I think that same rule applies in physical diagnosis—you can hear the low-pitched notes easier than the high-pitched, given notes of the same intensity.

Dr. Viko's paper was of particular interest to me, because of the frequency with which we meet this type of case. As he says, it has not been brought out in the medical literature, but we see these cases very frequently indeed. It was particularly hard during our examination of soldiers in France to determine sometimes just where one condition began and the other ended, and we see the same conditions, of course in civil life.

I do not feel qualified to discuss Dr. Sampson's paper. I would like to know his explanation of the cause of the murmurs. I am not quite clear in my mind why these murmurs were present at birth and were not demonstrable before birth. I think that is an interesting point and would like to know a little more about it.

DR. M. M. MYERS, DES MOINES, IOWA.—The treatment of rheumatic heart disease is a matter of much interest to physicians. Since former methods of treatment are not successful in either prevention or cure we should reach out for other therapeutic means. If it is correct that a dormant or low grade activity of the processes is continually present in the heart from the time of the primary rheumatic disease on through the years, it will be necessary to employ a method of treatment which will stay or completely stop this activity, and in that way prevent the cardiac breakdown which may not come until ten or fifteen years after the initial rheumatic infection.

It has been suggested that sodium salicylate, a drug of known effectiveness in acute rheumatic fever, if used in daily doses over long periods of time may be of additional assistance in the treatment. And with the thought that this drug may check the activity in the heart I have been using it in this way for several months. If others here have had experience with sodium salicylate used in this manner I would be pleased to know about it.

There is another point, not related directly to the theme of the papers read today. It concerns the use of the expression "leakage of the heart." This term was mentioned once or twice and the fact that it finds place in such a program as this indicates that our nomenclature needs revision. To tell a patient that he has "leakage of the heart" may induce cardiac neurosis because of fear created by an entirely erroneous conception regarding the state of his heart. One may say to the patient that the "function of the valves is disturbed" or that "there is a change from the normal condition of the valves," but we should discard the term "leakage."

DR. JOHN PHILLIPS, CLEVELAND, OHIO.—There are several points that I would like to mention in regard to the first paper. One interesting feature of the advanced cases of rheumatic heart diseases in children at the age of puberty is the tendency to infantilism, with a retarded development of the secondary sex characteristics. In improving the nutrition of these cardiac patients, after the acute stage of the illness has passed, and the temperature has been normal for three or four weeks, massage is very helpful. It should be started gently at first with passive movements, but later resisted movements may be used.

I have been very much impressed with the variation in the incidence of rheumatic

heart disease in patients in private practice as compared to patients in out-patient departments of a hospital. It is much more frequent in the latter group. I believe that the low incidence in private practice is due to the fact that these patients are not so often allowed to harbor foci of infection.

In contrast to the two previous speakers, my opinion is that syphilitic myocarditis is comparatively rare. Syphilis usually attacks the aorta, and the changes in the heart are secondary to the aortic lesion.

In regard to the prognosis in hypertensive cardiac disease my experience has been the same as Dr. Roberts'. In a recent analysis of my own cases, of those whose diastolic pressure was 130 or over, approximately 55 per cent were dead within two years. The prognosis in cardiac decompensation in rheumatic heart disease is much better in the laborer than it is in the man whose occupation is sedentary. In diseases of the thyroid, especially adenomata of the thyroid with toxic symptoms associated with arrhythmias of the heart and impaired myocardial function, the prognosis is much better after thyroidectomy.

It is very important in obese patients with arterial hypertension to reduce the weight by dietetic measures. Sometimes a marked drop in systolic pressure occurs coincident with the reduction in weight.

It is important to differentiate between psychoneurotic conditions with symptoms referable to the heart, and hyperthyroidism. Often the patients with the former condition have attacks at night of palpitation of the heart, disturbance of breathing and belching of gas. These have been described by Gowers as vasovagal attacks and are often associated with sexual irregularities.

DR. JAMES G. CARR, CHICAGO.—Our difficulty in Chicago and in Illinois has not been due to the unwillingness of business men or employers to accept the cardiac as an employee because of his infirmity or of the likelihood that he will often be absent from work. To a certain extent the prevalent idea that a cardiac is a candidate for sudden death has operated against his employment. A more effective reason, however, is the fact that our state law does not permit a prospective employee to sign a waiver of liability on the part of the employer for disabilities which may arise from the cardiac disease. The employer must accept the employee with the full responsibility imposed by the law and this fact makes the employer circumspect about accepting a man with a known disease, any later developments of which are apt to be attributed to his work. The next step in our campaign to provide employment for the cardiac is to secure some modification of the law regarding the validity of waivers.

I was particularly interested in what Dr. Musser had to say about the pathology of cardiovascular syphilis. At the Cook County Hospital our pathologists take the position that cardiac syphilis is essentially vascular. Not long since in a discussion with the pathologist then serving as our resident I insisted that other pathologists accept the diagnosis of syphilitic myocarditis as valid and met with the reply: "I do not deny that syphilitic myocarditis does occur but we rarely find it. The cardiac changes in syphilis are almost invariably secondary to vascular disease of the aorta or coronary vessels." We meet an enormous amount of cardiac syphilis at the County Hospital and are especially interested in the subject. I believe one factor in the unfavorable prognosis usually associated with this condition is the result of the aortic regurgitation which is so often present when the patient comes under observation. The associated low diastolic pressure is responsible for poor circulation within the coronary vessels, and the muscle is insufficiently nourished even in the absence of vascular or inflammatory disease.

I was also interested in what Dr. Musser had to say about the treatment of cardiac syphilis. We are willing to admit that the question has not yet been settled.

We are careful about the use of arsenicals in this condition. We have used neosalvarsan in these cases though not in patients with decompensation. In a certain number of cases we have secured good results. On the other hand, some of our patients have not done well under such treatment. Within a year one patient who first came to us with a minor grade of decompensation was given, after compensation was restored, a course of neosalvarsan. After a few doses he began to lose ground and became decompensated again; from this second attack of decompensation he did not recover. We feel that many questions in connection with cardiac syphilis are still unsettled. Our present methods of treatment are not satisfactory, and much information is needed as to the indications and contraindications for the use of specific therapy in these conditions.

DR. G. WERLY, EL PASO, TEXAS.—I am encouraged to make a few remarks by what Dr. Roberts said about the practitioner in small towns.

I was interested in Dr. Horine's paper on prognosis. I was intensely interested in prognosis in heart disease five years ago, when I had an attack of cardiac infarct myself. I think he should have added another paragraph on treatment. I think we can do a great deal for people who are very sick with heart disease some times. My case was rather badly managed, because I managed it myself. I only rested ten days and then gradually went back to work and after I got back to work I took careful exercise, and then more and more, and now after five years I can go up a mountain 5,000 to 7,000 feet very well.

I think in these cases we make a great mistake in not considering the pathology with which we have to deal. I read all the papers I can find on this subject by you gentlemen, but I find you do not say much about rest, and of all things that a heart damaged by cardiac infarct needs, it is rest, and then more rest. I have a case that I have had in bed for thirteen weeks, and now I am letting him up gradually. Other cases I have kept in bed a month and longer. Then I allow them to be up and around, but I insist they shall do no work for six months more. I have noticed in my own case that soon after the attack I had pain in the chest on walking, and especially pain after meals. Nearly all of us eat too much and these cases need instruction about how much to eat. But year after year, I have been better and now I hardly realize that I ever had an attack.

DR. GEORGE L. CARLISLE, DALLAS.—I would like to say a word about a point in which I am particularly interested. I am not a cardiologist; I am not a specialist in heart disease, but it seems to be more and more apparent that a cardiac disease should be classified so clearly from an etiologic standpoint that rheumatic heart or a streptococcic heart could not possibly be mistaken for anything else.

I am of the opinion, simply from observation of these cases clinically, that perhaps most of this infection starts in the throat. It may go to the sinuses or elsewhere, but it starts in the throat, and principally in the tonsils. I was interested in Dr. Stulik's statement that about 70 per cent of these tonsils were incompletely removed. Every case in which the tonsils are attacked surgically is not successfully completed, and bits of tonsils are just as dangerous to the patient as two bad tonsils. I believe the tonsils should be removed routinely at four years, provided there is no contraindication in children. You must stop this trouble before it enters the circulation, because when the streptococcus becomes implanted you cannot remove it.

I have one other thought in mind. As internists we see these cases in the hands of specialists of various kinds. I believe that the cardiac man should realize that the psychiatrist is just as good a man in his field as the cardiologist is in his, and because the cardiologist cannot relieve a functional cardiac neurosis is no reason why it cannot be relieved. I believe in many of these cases a good psychiatrist can

get at the condition, can find the basis of the trouble and put the patients back on a profitable basis to themselves and their families.

I want to express my appreciation at hearing this program. I was a little nervous when I received the invitation to preside at the American Heart Association, but it has been a very enjoyable meeting. We will now give these gentlemen a chance to defend themselves.

DR. R. H. OPPENHEIMER (closing).—I have nothing to say except to thank the Association for the privilege of being here.

DR. EMMET F. HORINE (closing).—I think all of us are more or less familiar with Warthin's work in finding the spirochetes in the heart muscle. This was in 1916. And even without the ability or the patience of Warthin many other workers have been able to demonstrate the spirochetes in the heart muscle. Prior to Warthin's work, Reuta, Benda and also Schmorl as early as 1906 and 1907 demonstrated spirochetes in the aortic wall. However, the question I would like to ask is this: Has it been demonstrated that the actual presence of the spirochetes in the heart muscle produces myocardial failure? Is it not true that the involvement of the aorta itself plus the thickening of the coronary lumina and reduction of the blood supply to the myocardium is the probable cause? In addition to the actual narrowing of the coronary lumina we have more or less perivascular infiltration present.

The subject of treatment was only mentioned in a general way. I agree with the statement made by Dr. Musser concerning the efficacy of bismuth. I think the treatment of cardiovascular syphilis should be based on mercury and the iodides and bismuth. To my mind the arsenicals are definitely dangerous in the later stages of cardiovascular syphilis. The time to treat these cases in that way is in the primary stage, but when there is involvement of the aorta or an aortic insufficiency present, I do not believe the arsenicals should even be seriously considered.

There must be some difference in the way patients behave in Atlanta and Cleveland as compared to Louisville. Is it that we are more fortunate in securing better cooperation from our patients and thus they carry on for a longer period? While I concede that a diastolic level at 120 does carry with it a high ultimate mortality still it does not carry with it necessarily an early death. I have had opportunity to follow a group of cases with this diastolic level over several years, some even as long as ten, and they are still in good condition. The higher the diastolic level is constantly maintained, however, the greater the danger and, as I mentioned in the essay, when at 150 or above there is certainly a probability of an early death.

I do agree most fully with what Dr. Myers said concerning the necessity for discarding the term "leakage of the heart." The earlier we adopt an etiological classification of heart disease, the better it will be. Then we will have something definite to consider in the way of treatment and certainly a more definite concept of the condition present.

DR. HOWARD B. SPRAGUE (closing).—I agree with Dr. Musser that a loud diastolic murmur is easier to hear, but a short diastolic murmur heard immediately after the third sound is difficult to hear. In the case of mitral stenosis with a loud presystolic roll, the systolic may be difficult to hear.

Dr. Robberts suggests that possibly living up where Dr. Cabot comes from we lose some of the illusions that they have in other parts of the country. As you remember Dr. Cabot in his recent book about the heart describes seven cases of that "rare clinical entity," *mitral regurgitation*. In looking up some of those cases my-

self I find he admits that one case died of mitral regurgitation that had a mitral valve 17½ cm. in circumference, almost twice the normal circumference. We find that clinically we have to admit "pure" regurgitation at times when a valve ring is much smaller than that. We really do not, however, make a diagnosis of organic mitral regurgitation very easily. In fact, we agree with Rothschild that you must have a rheumatic history and a murmur or enlargement and the murmur.

As far as transmission of murmurs is concerned, I feel it depends upon the intensity of the murmur and has nothing to do with its functional or organic quality. I quite agree with Dr. Roberts that the murmur should at least be transmitted to our ears.

Dr. Myers' question as to the use of the salicylates over long periods—we are beginning to feel that the use of the salicylates over long periods is a good idea, so in the past year we have been giving daily rations of aspirin—ten or fifteen grains—to children coming to the cardiac clinic during the fall, winter and spring. That idea was started by Dr. Emerson at the Children's Hospital in Boston.

Every case of mitral disease is not mitral stenosis. It usually is pathological, although once in a while even at necropsy we find mitral regurgitation without stenosis. It is important to differentiate *organic* regurgitation because I have seen cases in which subacute bacterial endocarditis was engrafted on hearts in which, before the reinfection, nothing could be heard at the apex but a loud systolic murmur. One case of rheumatic heart disease I remember, that had been watched for twelve years, in which for eight years only the apical systolic murmur was heard, but in the past three years the systolic murmur has entirely disappeared and a very loud diastolic murmur of mitral stenosis is the only murmur that can now be heard. At any time during the first eight years it is probable that the murmur would have been called a "functional" murmur, and in fact one or two of the children in our series of organic mitral regurgitation have been considered cases of functional systolic murmurs by Dr. Cabot.

DR. T. HOMER COFFEN (closing).—The answer to Dr. Musser's question as to what constitutes heart failure is considered in the paper but was not mentioned in the abstract. I had Mackenzie's definition of heart failure in mind: "That condition in which the heart is unable to maintain an efficient circulation during the efforts necessary for the daily life of the individual."

DR. LOUIS E. VIKO (closing).—With regard to the rôle of the psychiatrist in the treatment of cardiac neurosis, I agree with the doctor. Possibly every case of any uncomplicated cardiac neurosis should be referred to the psychiatrist after the cardiologist has ruled out organic disease. The cases referred to in my paper where organic lesion and neurosis coexisted, should probably, in most instances, be treated jointly by the psychiatrist and the cardiologist. The anxiety and introspection types, however, are usually easily relieved. It is in the treatment of the neurasthenic and hysterical types where the psychiatrist is of greatest value.

DR. J. J. SAMPSON (closing).—The question which Dr. Musser asked regarding the causation of murmurs before and after birth is not simple to answer. The murmurs that have been observed clinically before birth in hearts proved to be both normal and pathological, have been laid by previous observers both to variations in the currents in the fetal heart and to pressure on the umbilical cord. The findings in our work which suggest certain causative factors are that these murmurs occur frequently but not constantly, that they are associated with the characteristic sounds and therefore are probably of cardiac origin, and that in some cases they resemble

the murmurs observed in postpartum cases of patent ductus arteriosus and of patent foramen ovale. We are not able to do more than suggest the causes of the murmurs, excluding the case of pulmonic stenosis observed, but especially where the diastolic murmurs persist we may conjecture as to possible minor pathological structural changes. Clinically we have no grounds for assuming diagnostic significance other than in a negative capacity.

Regarding the murmurs heard after birth, the diastolics have been mentioned and the early systolics may be classed with the so-called functional murmurs which have been much discussed and I think most ably covered relative to etiology in the paper of Dr. Thayer last year. The explanation is still incomplete but the theory of a relative mitral insufficiency early in life due to a dilatation of the valve ring and ventricle owing to the added function thrust upon the latter at birth, seems rational.

Department of Reviews and Abstracts

Selected Abstracts

Cotton, Thomas F.: Modern Methods in the Diagnosis and Treatment of Heart Disease. Canadian Med. Assn. Jour., 1926, xvi, 487.

In this address the author reviews the methods which have been adopted by medical officers at the Ministry of Pensions. These methods he states are based on the principle that abnormal signs, when associated with perfect exercise tolerance are of little diagnostic value and of no prognostic importance, and conversely that symptoms of congestive failure are always accompanied by signs of structural disease.

It is of little importance what type of exercise test is employed. All serve the same purpose providing a normal standard is known by which undue symptoms of distress can be recognized. From the history alone and the appearance of the patient in many cases with structural disease the exercise tolerance can be determined. Rise in pulse rate, changes in blood pressure and increased respiration rate are fallacious signs when considered alone as tests of cardiac deficiency.

The signs of structural disease which matter are those which indicate failure with congestion or angina, cardiac enlargement, aortic dilatation, gross valvular disease, active infections, certain types of irregular heart action and the state of the arteries.

Yacoel, J., and Giroux, R.: Artificial Pneumopericardium in Pericarditis with Effusion. Arch. d. Mal. du Coeur, 1926, xix, 158.

The authors report great improvement following the injection of nitrogen into the pericardial sac of a patient suffering from tuberculous pericarditis with effusion. Death finally resulted from peritoneal tuberculosis.

Harmer, I. M.: On the Etiology of Aortic Regurgitation. Heart, 1926, xii, 371.

Four hundred and sixty-seven patients with aortic valvular disease out of a total of 2,560 patients attended the outpatient department at the University College Hospital from 1919 to May, 1925. These patients were all male pensioners from the Army. The following observations have been recorded by the author. Syphilis is an important factor in the production of aortic regurgitation. Thirty-seven and three-tenths per cent of all cases of aortic regurgitation between 20 and 70 showed a positive Wassermann reaction. Rheumatism is of rather less importance and accounts for 27.2 per cent.

Pure aortic regurgitation is mainly a disease of middle life, while combined aortic regurgitation and mitral stenosis is essentially a disease of young people.

Syphilis is of more importance as an etiological factor in patients over 35 years of age, thus syphilitic aortitis and hence aortic regurgitation is not pronounced enough to cause cardiovascular symptoms until 20 to 25 years have elapsed from the infection. Rheumatism is of more importance as an etiological factor in patients below 35 years of age because rheumatic fever is essentially a disease of young people and cardiovascular symptoms due to rheumatism appear on the average 11.6 years after the attack of rheumatic fever.

Cotton, T. F.: Cardioaortic Syphilis and Its Treatment. Brit. Med. Jour., 1926, i, 855.

A group of 200 patients with syphilitic aortitis were studied in the cardiac clinic at the University College Hospital. There was nothing in the symptoms or physical signs to distinguish these cases from another group under observation during a similar period with aortic disease of rheumatic origin. According to symptoms and signs, the author divides the cases into the following groups: (1) those with heart failure of the congestive type; (2) those with symptoms due to loss of cardiac reserve, but with the myocardium not sufficiently damaged to cause venous stasis; (3) a small group complained of anginal pain; (4) those with hypertension often but not always with albumin and casts in the urine; (5) those with signs of aortic disease but no symptoms of myocardial insufficiency.

An unselected group of 58 cases were chosen and given treatment under intensive observation along with an equal number of control cases who received no treatment. It is justifiable to conclude from these observations that specific treatment is indicated in syphilitic aortic disease, that no danger is attached to the intravenous injection of arsenic in moderately large doses given without any special precautions; and that arrest in the disease, as shown from the after histories of cases treated, may be due to specific treatment given over a period of 5 years.

Dana, H. W., and Dameshek, Wm.: Intermittent Auriculoventricular Block. Am. Jour. Med. Sc., 1926, clxxi, 485.

One case is reported of a man showing partial heart-block with a 2:1 rhythm which disappeared and reappeared repeatedly with normal cardiac rhythm in the interval. A polygraphic record shows the period of transition from block to normal rhythm. The P-R conduction time was longer during the period of normal rhythm. Also the patient experienced more discomfort during this period. In explanation of the possible mechanism of transient partial heart-block the authors agree with the opinion advanced by Carter and Dieuaide, that in these hearts the bundle of His contains a few intact fibers just equal to the work of conducting impulses normally under favorable conditions, but incapable of transmitting the stimuli whenever stresses of any sort occur.

Keith, Norman N., Smith, Florence H., and Whelan, Mary: The Therapeutic Use of Diets Low in Water and in Mineral Content. Arch. Int. Med., 1926, xxxvii, 550.

A calculated standard basal diet was worked out for use in patients with edema or ascites from various causes. The diet contained 40 grams of protein from eggs and milk, 400 gms. of fresh fruit and green vegetables made up to 600 gms. by the use of other fruits and vegetables and made up to 1500 calories by the addition of sugar and fats. The total fluid intake was 1400 to 1600 c.c. daily, 600 to 800 c.c. being actual fluid.

After the caloric requirement and the ability of the patient to use protein food had been determined, the diet was adjusted to meet the actual requirements of the individual. The attempt was made to hold the water and mineral content as nearly constant as possible while the protein and calories were increased.

This diet regime has been used in eight cases of subacute and chronic nephritis and twenty-five cases of ascites from liver disease or cardiac insufficiency. There have been no demonstrable ill effects from the continued use of these diets. The diets have been effective as shown by instantaneous diuresis, loss of edema and de-

crease of body weight. The patients' adherence to the diet is of interest for they seem to realize from experience that if the intake of water and salt is increased beyond their tolerance, an increased retention of fluid and a gain in weight occurs.

Swetlow, George I., and Schwartz, Sidney P.: The Treatment of Cardiac Pain by Paravertebral Alcohol Block. Jour. Am. Med. Assn., 1926, clxxxvi, 1679.

Five patients were relieved of severe cardiac pain by paravertebral injections of alcohol.

Constant pain stimulation of the small cells in the dorsal root ganglions by impulses coming from the heart is referred to the skin segments supplied by the respective ganglions which, in turn, refer this irritation to the consciousness as pain. The axones of these small cells are unmyelinated and form fibers which convey protopathic stimuli. The introduction of alcohol into these irritable nerve fibers produces a Wallerian degeneration. The site of the injection is determined by carefully mapping out skin areas of hyperesthesia, hyperalgesia and hyperthermesthesia on the chest wall. The nerve roots and dorsal root ganglions supply the areas of increased epicritic and protopathic sensibility and presumably the ones kept in a state of irritation by the bombardment of afferent impulses from the heart. With the usual technic of paravertebral alcohol injections from 3 to 5 c.c. of an 85 per cent solution of alcohol is injected into the nerve roots identified by the foregoing procedure.

Silbert, Samuel: The Treatment of Thrombo-Angiitis Obliterans. Jour. Am. Med. Assn., 1926, clxxxvi, 1759.

Sixty-six patients have been treated since 1923 by repeated intravenous injections of 5 per cent sodium chloride solution. The amounts used were 300 c.c.; the first injection of 150 c.c. at intervals of two days; later, twice weekly, and finally once monthly so long as the patient remains under observation. Twenty-five hundred injections in all have been given with no dangerous reactions or untoward results. During the injection the blood volume is increased and the patients often state that they feel warm; the face becomes flushed and the superficial veins engorged. They often complain of thirst. The salt solution produces a certain amount of blood destruction. In view of the high hemoglobin and red cell count, this is of no serious consequence. On theoretical grounds it would seem that the repeated increase in the blood volume necessarily stretching the vascular system is responsible for the improved circulation and the development of the collateral blood supply that results. Another possible factor is the dilution of the blood with the resulting decrease of viscosity.

Forty-six patients have been improved, 14 are unimproved or it is too early to judge results and 4 have resulted in amputations. Improvement consists of cessation of pain, increased temperature of the extremity, growth of the nails, healing of ulcers and reopening of obliterated vessels. The treatment must sometimes be continued for months.

Keefer, Chester S., and Resnik, William, H.: Jaundice Following Pulmonary Infarction in Patients with Myocardial Insufficiency. Jour. Clin. Invest., 1926, ii, 375.

The authors report ten cases of myocardial insufficiency in which the sudden appearance of physical jaundice or the sudden increase in jaundice that was already present seemed dependent on the development of pulmonary infarction. The jaundice was due in all instances to bilirubinemia.

In a group of animal experiments it was demonstrated that anoxemia may depress the function of damaged livers to such an extent that bilirubinemia appears. It was also shown that anoxemia failed to produce bilirubinemia when the liver was normal and when anoxemia, even though severe, was present for several hours only. Reasons are given for assuming that pulmonary infarction in patients with myocardial insufficiency produces anoxemia, probably severe in some cases.

Levine, Samuel A., and Matton, Marcel: Observations on a Case of Adams-Stokes Syndrome, Showing Ventricular Fibrillation and Asystole Lasting Five Minutes, with Recovery Following the Intracardiac Injection of Adrenalin. Heart, 1926, xii, 271.

A case of Adams-Stokes disease is reported in which attacks of syncope lasting from several seconds to about 5 minutes occurred. During the very long attacks adrenalin chloride was injected directly into the heart and was followed by prompt recovery. In one of these and for a period of 5 minutes no heart beat could be seen, heard or felt. It is suggested that under such circumstances the customary beneficial effects from adrenalin, if given subcutaneously cannot be obtained, as the circulation is then at a standstill. In one of the lesser attacks from which the patient recovered a ventricular fibrillation lasting 3½ minutes and followed by complete inactivity of the ventricle for 79 seconds was displayed. Electrocardiographic records of this ventricular standstill are included in the report. The patient died suddenly 2 months after this occurrence, presumably in a similar and untreated attack.

Danzer, C. S.: Retrograde Capillary Pulsations in Heart Failure. Am. Jour. Med. Sc., 1926, clxxi, 654.

A retrograde capillary pulsation coming from the venules back into the capillaries is described in a case of heart failure. The visualization was made by the Lombard-Mueller-Weiss method. There were many intervascular anastomoses and numerous capillaries of the convoluted variety. The venous portions were especially dilated. In one the corpuscles altered their course, taking first the normal then the reverse direction. In a number of other capillaries there were phases when the corpuscles coming from the arterial limbs of the capillaries collided with those regurgitated back from the venules to the capillaries, the latter was obviously the result of venous stasis and regurgitation attending the tricuspid insufficiency.

The pressure in the capillaries was determined with a microcapillary tonometer. As the pressure was increased the changes showed first a "beaded flow" falling directly to the 4th stage of compression which the author calls "corpuscular reversion."

The presence of a retrograde capillary pulse may be of value in the diagnosis of doubtful cases of tricuspid insufficiency or severe venous stasis.

Willius, Frederick A.: The Mode of Death in Various Types of Heart Disease. Am. Jour. Med. Sc., 1926, clxxi, 480.

An analysis of the nature of heart disease in a consecutive group of 330 cases is presented. The nature of death in this group is expressed. The types of heart disease were as follows: (1) arteriosclerotic cardiac disease, 25 per cent; (2) myocardial infarction, 11 per cent; (3) hypertensive cardiac disease, 12 per cent; (4) cardiac hypertrophy associated with obesity, 0.6 per cent; (5) acute myocarditis, 0.3 per cent; (6) cardiac failure from mechanical causes, 0.6 per cent; (7) chronic

endocardial valvular disease, 28 per cent; (8) bacterial endocarditis, 9 per cent; (9) pericardial disease, 5 per cent; (10) cardiovascular syphilis, 8 per cent; (11) congenital cardiac disease, 1 per cent. Sudden death occurred in 125 cases (38 per cent) while death from gradual cardiac failure occurred in 205 cases (62 per cent). Embolic phenomena occurred in 58 cases. Necropsy was performed in 85 per cent of the cases reported.

Gallavardin, L.: Terminal Ventricular Tachycardia. Arch. d. Mal. du Coeur, 1926, xix, 153.

The author describes four cases of ventricular tachycardia complicating fatal heart disease. Transitions were noted between isolated ventricular extrasystoles, bigeminal rhythm, showers of extrasystoles and longer paroxysms of ventricular tachycardia. In the latter transitions were observed between regular alternation and gross diversity of the ventricular complexes. The author believes all these ectopic ventricular rhythms are expressions of circus movements within the ventricles (ventricular flutter). The regular alternation of the ventricular complexes would be explained by alternation of the pathways of the circus movement and when the latter become less uniform, multiform complexes result. The author states that it is possible to isolate from the vast group of extrasystolic arrhythmias a severe form of ventricular extrasystolic rhythm accompanying grave cardiopathies and exhibiting a special tendency to bigeminy (either spontaneously or under the influence of digitalis) and to extrasystoles first in the form of brief showers and later of more prolonged paroxysms with the permanent danger of fatal ventricular fibrillation.

Doumer, E.: Total Bradycardia with Disturbances of Conduction and Transitory Extrasystolic Bigeminy during the Course of an Attack of Acute Articular Rheumatism. Arch. d. Mal. du Coeur, 1926, xix, 33.

During the course of a recurrent attack of acute rheumatic fever, the patient developed a rapid irregular pulse for which she was given 5 drops of a 1:1000 solution of digitalin for 5 days. On the sixth day, her pulse was 32 and the rhythm coupled. A short time later, the bigeminy was replaced by sinus bradycardia with prolongation of the P-R interval, but the former rhythm could be made to reappear by deep respiration or exercise and later by ocular compression. The patient was finally discharged with a regular pulse of 52. The author believes that the small amount of digitalis administered was not responsible for the disturbances of rhythm. Inasmuch as the electrocardiographic changes (sinus bradycardia, prolongation of auriculoventricular conduction, escaped extrasystoles) are those produced by vagal stimulation, he concludes that the rheumatic interstitial nodules affected the cardiac nerves more than the myocardium itself.

Crawford, J. Hamilton, and Rosenberger, Heinz: Studies on Human Capillaries: I. An Apparatus for Cinematographic Observation of Human Capillaries. II Observations on the Capillary Circulation in Normal Subjects. III. Observations in Cases of Auricular Fibrillation. Jour. Clin. Invest., 1926, ii, 343, 351, 365.

An apparatus is described by means of which it has been possible to take cinematographic records of the capillaries at the nail fold in the human subject.

The observations show that the diameter of the capillaries is constantly changing, although the individual capillary does not appear and disappear as they do in certain other tissues, nevertheless, definite variations occur in this situation. They also

show that the reactions which take place are of a similar nature over a period of several days and that different subjects exhibit individual peculiarities in the changes seen.

The width of the blood stream has been used as a criterion of the capillary size because they remain constantly full of blood except for the occurrence occasionally of small gaps in the stream. Observations show that there is no zone of pure plasma surrounding the axial stream of corpuscles.

The extent of the changes in the arterial and venous limbs of the same capillary were about the same, but the various capillaries in the same subject differed from one another. Marked variations were also seen between the different subjects. There was no evidence that these were due to a peristaltic wave of contraction, a contractile motion of the capillary, like that of the heart, or a pulsatile motion conveyed to the blood stream by the heart beat. The mechanism of their production is uncertain.

The caliber of the arterial and venous limbs of the capillaries has been studied in such cases of auricular fibrillation both before and after digitalization. The extent of the changes during the stage of decompensation was much greater than had been observed in normal subjects, but when compensation became established under digitalis the variations became less marked. The magnitude of these changes was dependent upon the state of cardiac decompensation and had no relation to the rate or amount of irregularity of the pulse. In cases of marked decompensation the stream was small in most of the capillaries and had a granular appearance while in many stasis was present.

Long, Perrin H.: Effect of Phenylhydrazine Derivatives in the Treatment of Polycythemia. Jour. Clin. Invest., 1926, ii, 315.

Phenylhydrazine hydrochloride and acetyl phenylhydrazine are efficient therapeutic agents in the treatment of polycythemia vera. In this condition with no discomfort to the patient the blood can be reduced to normal and held there with ease for a considerable period of time. With a normal blood picture these patients lose the symptoms of their polycythemia and are greatly benefited. No patient was seen in whom any signs of liver damage arising from phenylhydrazine could be detected.

Small doses of the drug in the beginning of the treatment were used. One-tenth gm. per day by mouth, continued until the red blood cell count falls or until 1 gm. of the drug has been given. Then the drug should be discontinued for 3 days. If there is no significant change in the blood picture the drug should be given again, alternating 0.1 gm. and 0.2 gm. doses until the desired results have been obtained or until another gram has been administered. The red blood cell count, hemoglobin and white blood cell count should be determined daily in order that any sudden change in the blood picture may be detected at once.

The drug when taken by mouth does not cause irritation of the gastrointestinal tract. It was absorbed into the blood and caused methemoglobin to be formed. This apparently injured the red blood cells and allowed them to be phagocytized and then destroyed. The white blood count did not seem to be affected and the differential count remained normal under this treatment. Reticulated red blood cells increased as the total count dropped. The platelets remained normal. The patients developed slight jaundice and the blood bilirubin was increased in amount and gave the delayed direct diazo reaction. One patient with polycythemia vera was treated with satisfactory results. Later the condition recurred with improvement when treatment was recommenced. Other patients with polycythemia due to other causes show a reduction in the polycythemia but no improvement in their general condition.

Wedd, A. M.: Veratrum Viride in Auricular Fibrillation. Heart, 1926, xii, 307.

The action of veratrum viride given orally has been studied in 20 cases of clinical auricular fibrillation. There were produced a slowing of both the auricular and ventricular rates of beating and a fall in blood pressure. These changes were independent of general toxic effects.

Veratrum viride has, in addition to the vagal action already observed, a direct action similar to quinidine upon the auricular muscle of the dog.

The slowing of the ventricular rate by veratrum given orally occurs much earlier than that following digitalis bodies. However, as a therapeutic agent veratrum is somewhat handicapped by uncertainty and irregularity of action. It may be active in cases in which simultaneous slowing of ventricular rate and lowering of blood pressure is desired.

Otto, Harold L., and Gold, Harry: The Effect of Digitalis on Ventricular Premature Contractions. Arch. Int. Med., 1926, xxxvii, 562.

In a patient with arteriosclerosis, hypertension or enlarged heart, premature ventricular contractions were completely abolished by digitalis given in full doses. After the premature contractions were abolished they were prevented from recurring by maintenance doses of 3 grains of the dried leaf.

Cattell, McKeen: Observation on the Action of Digitalis on the Frog Heart and Its Modification by Quinidine. Jour. Pharm. and Exper. Therap., 1926, xxvii, 287.

Isolated frog hearts were perfused by the Straub method. Kymograph records were obtained by means of a heart lever attached to the apex of the ventricle. A tincture of digitalis assaying 0.007 c.c. per gram of frog, diluted 50 times with Ringer's solution was used and would stop the heart in about 10 minutes. From the records obtained two measurements were made: (a) the period elapsing before the amplitude of contraction is reduced to one-half the initial amplitude and (b) the time taken to bring the heart to a standstill. In control experiments, using digitalis alone, the period (a) required on an average 7.5 minutes while 10.6 minutes were required before complete block was developed. When quinidine sulphate in a strength of M/25,000 and M/50,000 of the alkaloid was added to the digitalis solution, no change was noted in the time required for the development of complete block. However on the addition of quinidine to the perfusion fluid systole was only partial at the point on the record where block became complete; the alternate contraction which continued showed a greater amplitude commonly as great as one-half the normal than they did with digitalis alone, or they continue for a considerably longer time.

When the heart was perfused for 8 minutes with the quinidine sulphate solution previous to the digitalis perfusion the time interval (a) was prolonged on the average to 11.6 minutes and the interval (b) was delayed to 19.6 minutes. When a mixed solution of the digitalis and quinidine was perfused after 8 minutes perfusion with the quinidine solution alone, the combination showed a slightly greater effect of quinidine in delaying the toxic effects of digitalis.

When weaker solutions of digitalis were used the characteristic systolic standstill could, in some instances, be entirely prevented by the use of quinidine. A heart poisoned by digitalis so that the ventricular contractions had practically ceased may be revived more rapidly with a quinidine solution than with Ringer's solution alone.

Dreyer, N. B.: Some Effects of Anoxemia on the Circulation. Canadian Med. Assn. Jour., 1926, xvi, 26.

Using decerebrate cats, the author studied the effect of varying degrees of anoxemia on the appearance of Traube-Hering curves in the arterial pressure. When anoxemia was induced the arterial pressure and respiration were unaffected for some time. After ten or fifteen minutes some deepening in respiration occurred until after another fifteen to twenty minutes a relatively constant level was reached. The general level of the arterial pressure showed a slight tendency to fall if anoxemia was prolonged. When the anoxemia became sufficient to give rise to cyanosis of the mucous membranes, Traube-Hering waves almost invariably made their appearance. If the degree of anoxemia could be kept comparatively constant, the Troube-Hering waves persisted almost indefinitely. Determination of the oxygen saturation of blood in two sets of experiments showed 20.9 vols. of O_2 per 100 c.c. of blood in the beginning, 15 vols. two and one-half hours and 12.6 volumes another hour after anoxemia was induced.

Sufficient carbon dioxide was introduced in the inspired air to bring about fairly severe dyspnea. The arterial pressure showed no Traube waves. It would seem, therefore, that the Traube-Hering curves are primarily due to an inadequate supply of oxygen to the vasomotor center and that they are in no way dependent on changes in the activity of the respiratory center.

Danielopolu and Proca: Observations upon Atrioventricular Rhythm in Man. Arch. d. Mal. du Coeur, 1926, xix, 217, 225, 232, 241, 247.

The authors, in a comprehensive study of two cases of atrioventricular (A-V) rhythm, emphasize the importance for the diagnosis of making numerous electrocardiographic tracings over a long period of time and of supplementing the study of attempts to modify the rhythm by vagal and ocular compression and by certain drugs. A negative P-wave should arouse suspicion of A-V rhythm, but occurs also in some cases of sinus rhythm. The P-wave in nodal rhythm may be negative, isoelectric, diphasic, polyphasic and occasionally even positive. The P-wave may be negative in one derivation and positive or polyphasic in another. The centers in the A-V region are frequently divided into superior nodal, middle nodal and inferior nodal, but in reality there are many more foci within the node and the position of the excitatory center in the latter determines the shape and position of the P-wave and the duration of the P-R interval.

The variability in the same patient of the form of the auricular complex is one of the most important signs of atrioventricular rhythm especially if there is inequality in the P-R intervals. Thus a negative P-wave and shortened P-R time in Lead III may be noted at one time and a positive P-wave and normal P-R time at another. In still other tracings taken from the same patient a negative P-wave may follow the R-wave. In the authors' second case there were two P-waves, one before the R-wave and the other after it. It appears that in this instance the impulse initiated in the A-V node reached by different paths two portions of auricle separated from each other by a region of block.

With respect to the diagnostic importance of the relative positions of the P-wave and QRS complex the following observations were made: (1) In single tracings the normal or altered P-wave may precede the R-wave, the P-R interval being shortened or even normal. In such cases the diagnosis may be difficult unless a number of tracings are made. (2) The P-wave may coincide with the R-wave in which instance the diagnosis is more certain. The chief possibility of confusion would be in cases of simultaneous independent contractions of auricles and ventricles. (3) The nega-

tive P-wave may follow the R-wave in which case too the diagnosis of nodal rhythm is highly probable. Similar findings are noted in occasional cases of independent auricular and ventricular rhythm or of idioventricular contractions transmitted to the auricle by a retrograde path.

The following classification of atrioventricular rhythm is given by the authors:

- A-V Rhythm
  - Transitory A-V Rhythm
    - Paroxysmal form (paroxysmal A-V tachycardia).
    - Nonparoxysmal forms
      1. A-V rhythm provoked by purely nervous influences.
      2. Transitory A-V rhythm in infectious diseases and certain chronic affections.
  - Permanent A-V Rhythm
    - Bradycardial form
      - Simple variety.
      - Complex variety.
    - Tachycardial form
      - Simple variety.
      - Complex variety.

The bradycardial form of persistent nodal rhythm is far more frequent than the tachycardial form. One or more of the signs enumerated above acquires great diagnostic value when the rate falls to 40, or so. In the complex varieties of nodal rhythm, various myocardial lesions modify the electrocardiographic findings. The authors mention, without giving details, a case of simple nodal tachycardia, which has persisted since 1914. In their second case flutter appeared during the course of an atrioventricular bradycardia of the complex type with the R-wave situated between two auricular complexes. The nodal rhythm persisted, but became tachycardial. For some time this rhythm stood in relation of 1:2 to the flutter, but later the two rhythms became independent of each other.

Any factor which abolishes the automaticity of the S-A node or exaggerates the irritability of the A-V node can precipitate nodal rhythm. In the first case, there was slowing of the sinus rhythm; in the second, acceleration of A-V rhythm. The most favorable conditions for the development of nodal rhythm obtain when both factors are operative at once. Permanent nodal bradycardia is due either to destruction of the S-A node or to sinoauricular block. In the rare cases of persistent nodal tachycardia, there is an irritative lesion of the A-V centers which function with such rapid rhythm that the sinus node can no longer control the heart beat. In such cases, there may also be destruction of the sinus node or sinoauricular block, but not necessarily so. The permanent forms of nodal rhythm are dependent on definitive lesions and not on neurogenic factors as in the transitory cases. Stimulation of the cardiac nerves can, however, shift the ectopic center of contraction from one part of the A-V node to another.

In the cases reported, the nodal rhythm was accompanied for a time by nodal extrasystoles and by flutter. In the second case, the flutter was partially due apparently to intraauricular block, one part of the auricle being in a state of flutter and the other contracting normally.

Compression of the vagus in the neck in nodal rhythm diminishes conduction in the A-V node, and causes shifting of the site of impulse production within the latter with resulting variability in the auricular complexes. Theoretically ocular compression should give similar results, but at times sympathetic effects predominate. In the first case, the rhythm became accelerated and the P-wave was displaced to a position after the R-wave. In the second case, atropine produced a positive dromotropic effect. Adrenalin produced acceleration of the nodal rhythm, excitation of a large number of ectopic foci within the node, a positive dromotropic effect upon direct and retrograde conduction, and an excitation of ectopic ventricular centers and even a short paroxysm of ventricular tachycardia. Digitalis caused the P-wave to fall

after the R-wave, acting by displacing the ectopic nodal focus and by diminishing retrograde conduction. Deglutition, forced respiration or exercise diminished both direct and retrograde conductivity.

Damez, Marie K., Dawson, Percy M., Mathis, Dorothy, and Murray, Margaret: Cardiovascular Reactions in Athletic and Nonathletic Girls. Jour. Am. Med. Assn., 1926, clxxxvi, 1420.

Two hundred high school girls were observed and were divided into athletic and nonathletic groups. The cardiovascular reactions of these groups were compared by the Schneider and Crampton tests for cardiovascular efficiency. The athletic girl differs but little from her nonathletic sister, as shown by the increase of pulse pressure on standing, the rise of diastolic pressure under like circumstances, the rise in systolic pressure and the fall in diastolic pressure found after exercise and the values of pulse product whether lying, standing or after exercise. On the other hand the resting pulse is slower, the pulse pressure larger in the athletic group. On rising from recumbency the pulse quickens less, it is less after exercise and it returns more promptly to normal. The Schneider rating is not less than 6 and the Crampton percentage is not less than 55 in the athletic group.

Alvarez, Walter C., and Zimmermann, Arnold: Blood Pressure in Women as Influenced by the Sexual Organs. Arch. Int. Med., 1926, xxxvii, 597.

The office records of 1,230 women entirely from the upper and middle classes form the basis of this study. The methods of analysis and the results of examination are outlined by the authors. The results are expressed as follows:

The average pressure for all ages in women who seem to be sexually normal is 134 mm. of mercury. The increase by decades is about 10 mm. for each period. Well proportioned women have systolic pressures that average 10 mm. higher than those of the thin and pressures in the stout average 12 mm. higher than in the well proportioned.

The sexually abnormal have pressures that average considerably higher. As the sexually abnormal tend to get married a little late, they find that single women have pressures averaging a little higher than those of the married women.

A masculine distribution of body hair, sex anesthesia, fibroids of the uterus, thyroid disease or pelvic conditions requiring ovariectomy and hysterectomy are associated with high average pressures. Abnormal menstruation, early menopause and pregnancy have no demonstrable effect.

Marked hypertension was found in a few girls who seemed to be normal sexually, but in these cases it was not of the primary type but was associated with severe forms of nephritis.

Mallory, William J.: Splanchnic Dyspepsia. Am. Jour. Med. Sc., 1926, clxxi, 504.

The author draws attention to a syndrome occurring in asthenic patients and referred to by them as "indigestion." These symptoms depend not upon changes in the gastrointestinal tract but to a disorder of the circulation. The real character is indicated by vasomotor disturbance, imbalance of sympathetic nervous system and a low Crampton's index.

Fossier, A. E.: The Cause of Essential Hypotension. Am. Jour. Med. Sc., 1926, clxxi, 496.

The following cardiovascular characteristics are found in essential hypotension: (1) The arch of the aorta is pulled down, its hemicircle is small and the size of

the transverse diameter over the area of the great blood vessels varies from 3.5 to 4.5 cm.

(2) The right border of the superior Vena cava is straight or sometimes slightly concave.

(3) The heart is vertical and poorly supported, and its transverse diameter is small and together with the great vessels appears drawn out and greatly elongated.

These characteristics are usually found in individuals of asthenic body type or with splanchnoptosis.

These individuals can be treated satisfactorily in two ways:

(1) With the use of an abdominal support, and

(2) By developing abdominal muscles and increasing intraabdominal fat.

Barnes, Arlie R.: Cerebral Manifestations of Paroxysmal Tachycardia. Am. Jour. Med. Sc., 1926, clxxi, 489.

Cerebral manifestations consisting of vertigo, hemianopsia, temporary blindness, fainting, falling with or without loss of consciousness, and epileptiform seizures occurred in 15 of the 104 cases of paroxysmal tachycardia that were studied. The presence of these symptoms requires that paroxysmal tachycardia be considered as a cause.

Cerebral anemia seems the most reasonable explanation for these manifestations. A moderately slow pulse rate is essential for normal filling of the ventricles. At the excessively rapid rate the filling and hence the output of the ventricles is below normal. This results in a fall in blood pressure. Whether symptoms indicating cerebral anemia will be present in a given case probably depends upon various factors.

Cerebral manifestations occurred most frequently in cases in which the origin of the impulse and its propagation in the heart varied the greatest from normal.

The presence of these manifestations does not affect the prognosis in attacks. The prognosis depends on the type and degree of the underlying cardiac damage.

Hamman, Louis: The Symptoms of Coronary Occlusion. Bull. Johns Hopkins Hosp., 1926, xxxviii, 273.

A scholarly presentation of the clinical features of a common condition which has received recently deserved attention. Every feature of the condition is fully discussed in this presentation. Reference to the literature is complete.

Lowenfeld, Margaret F.: On the Control of Heart Disease in Children. Brit. Med. Jour., 1926, i, 817.

The results of the first two years' work in the cardiac clinic at the Royal Hospital for Sick Children, Glasgow, show that of 93 cases dealt with, 5 recovered, 1 was improved, 2 developed a cardiac lesion, 1 was disabled and later recovered and 70 remained in status quo; the remainder were not tabulated. The observations of the children indicate that adequate rest in bed during the acute stage with limited exercise and a gradual return to ordinary conditions of living afford the best chances for recovery. The special clinic is necessary to direct the care of the children during the quiescent stage of the infection.

Book Reviews

FACTS ON THE HEART.—By Richard C. Cabot, M.D. W. B. Saunders & Company, Philadelphia and London, 1926. Pp. 781.

This book, of nearly 800 pages, is based upon the clinical and postmortem study of 1,906 patients who came to autopsy at the Massachusetts General Hospital and is, we believe, as Dr. Cabot states, the only book on the heart which is based entirely upon the observation of patients in whom the clinical findings were invariably checked by the pathological ones.

The work consists of 12 chapters and a final summary, and Dr. Cabot suggests that most readers will profit by reading the introductory chapter, which discusses the frequency of various forms of cardiac disease, the final summary, the summaries in the course of each chapter and such case histories as seem interesting. This advice seems to us sound, for in this way the important conclusions are obtained without wading through a sea of case records which, however, are in themselves worthy of perusal as occasion demands.

In the first chapter the author points out that nearly half of all individuals autopsied at the Massachusetts General Hospital in the past quarter century had suffered from some cardiovascular disease. He further states that a cardiovascular lesion is an important factor in 38 per cent of all deaths. He emphasizes the importance of the degenerative heart disease of later life and the chronic infective (rheumatic and syphilitic) processes as contrasted with the much less frequent acute and subacute infections and the congenital lesions.

The second chapter deals with rheumatic heart disease and the author stresses the almost unique etiological significance of the rheumatic group of infections in chronic mitral disease, the predominance of females as subjects of pure mitral disease, the compatibility of mitral disease with long life, the frequency of intracardiac thrombosis in decompensated and fibrillating mitral cases and the great rarity of mitral regurgitation as a valve lesion. He discusses the clinical differences between pure mitral disease and various combined valve lesions and then considers aortic regurgitation and stenosis, mitral regurgitation and fatal chorea.

Chapter III deals with syphilitic heart disease and illustrates by case records the anginal, aneurysmal and aortic valvular types. The long period of latency between the original infections and the development of symptoms, the great preponderance among males, the comparatively rapid progression to a fatal ending when symptoms appear, the value of the Wassermann reaction and the x-ray in diagnosis, and the infrequency of concomitant rheumatic infection are the important points stressed.

Chapter IV discusses hypertensive cardiovascular disease and the usually concomitant cardiac hypertrophy and dilatation, the commonest of all heart lesions. This chapter also discusses the cardiac changes associated with nephritis. The author comments on our

ignorance regarding the relationship of arteriosclerosis and nephritis to hypertensive heart disease, its more frequent occurrence in males, the lack of evidence of alcohol and muscle work in the etiology and the long duration of latency.

Chapter V discusses true fibrous myocarditis, its lack of a definite clinical symptomatology, the lack of evidence of its syphilitic nature, its rarity as the sole cardiac pathology, and its relation to coronary thrombosis. The chapter contains a discussion of the results of coronary closure and its symptomatology, and brief consideration of myocardial abscess, cardiac aneurysm and rupture of the heart.

Chapter VI discusses angina pectoris. The author strikes a decidedly pessimistic note when he opines that the very nature of the disease makes the solution of its exact etiology improbable. He states that his study of 138 cases throws no new light on the origin of the syndrome. He suggests the importance of clinically separating the symptoms of coronary occlusion from those of true angina. He emphasizes the syphilitic origin of about 16 per cent of the cases but states that in only a small proportion of these are the orifices of the coronary arteries obstructed.

Chapter VII discusses acute and subacute endocarditis. The author stresses the frequency of implantation of the infection on chronically diseased valves, the frequency with which embolic phenomena are missed clinically, the common incidence of an accompanying nephritis, and the not infrequent recovery of the patient, particularly in childhood.

Chapter VIII is concerned with nondeforming valvular sclerosis and emphasizes its clinical insignificance.

Chapter IX covers acute pericarditis. It emphasizes the much greater frequency in males, the frequent lack of pain as a pronounced symptom, the absence of fever in terminal cases, the difficulty of differentiating endocardial from pericardial murmurs, and the frequency with which the condition is overlooked.

Chapter X deals with chronic pericarditis. It stresses the importance of concurrent mediastinitis as a cause of serious cardiac failure.

Chapter XI deals with thyrocardiac disease, the over-demonstrative heart. The author states that death in such cases is usually toxic. He does not emphasize the relative frequency of auricular fibrillation in these cases.

Chapter XII deals with congenital cardiac disease and emphasizes the infrequency with which the condition is of practical significance.

The work is an example of the statistical method applied to the study of heart disease. It possesses the advantages and suffers from the defects inherent in the method. The method is, of course, an old one which, in recent years, has fallen more or less into disuse, probably too much so. It is the method of Louis and other distinguished clinical pathologists. In recent years it has been overshadowed by the experimental method. Both have their uses and there is little question that there is great advantage in restudying old problems in the light of new knowledge, by the statistical method.

The method suffers from defects, especially in the study of heart disease. There is considerable opportunity for diversity of opinion among competent clinicians both in the observation and the interpretation of cardiac signs, especially heart murmurs. Dr. Cabot realizes this difficulty and comments on it when he calls attention to the fact

that the clinical observations on his patients were made by a variety of clinicians. The pathological study of diseased hearts is likewise more difficult than is the case with most other organs, because the heart is a hollow muscle and, therefore, the condition of the orifices is to some extent dependent upon the state of contraction of the muscular tissue. For this reason it is not infrequently impossible to determine on the autopsy table just what conditions existed during life. Again, it is to be noted that conclusions based upon the observations of a single individual or the records of a single hospital suffer in accuracy from the comparatively small number of observations and this, of course, is most apt to be the case in connection with the less common diseases.

Certain of Dr. Cabot's views are so divergent from current opinion as to warrant special mention, particularly his comments on myocarditis and mitral regurgitation.

It is certainly true that the term myocarditis has been used in a most careless and unscientific manner. When that obsolete but orotund phrase "fatty degeneration of the heart" or "fatty heart" went out of fashion as a diagnosis, myocarditis took its place. As any pathologist knows, actual fibrous myocarditis of clinically significant extent is decidedly unusual. And yet it does not help matters very much to substitute hypertensive cardiovascular disease, the exact nature of which is also unknown. We must admit, however, that the latter term is several shades more accurate.

With regard to mitral regurgitation, there is much to commend in Dr. Cabot's views. It has been far too usual in the past to interpret almost any systolic apical murmur as evidence of mitral regurgitation, a view which is as full of fallacies as the proverbial egg is of meat. And yet, in view of the frequency of mitral stenosis, mitral regurgitation cannot be a rare disease. It is obvious to anyone who has studied the stiffened, retracted and often fused valves of mitral stenosis that they must, in most cases, be incompetent. It is probably true that in many instances the signs of stenosis mask the signs of insufficiency just as the opposite situation is true in connection with the aortic valve. We are inclined to think, however, that, on the whole, even though Dr. Cabot has logically based his inferences on the evidence, his statements regarding the infrequency of mitral regurgitation are too radical.

There are many other points in the work which are perhaps open to a somewhat different interpretation than those of the author but, of necessity, they cannot all be considered. All of Dr. Cabot's utterances, whether written or spoken, provoke thought, and, after all, this is one of the most useful and important functions of any book. We believe that if read with due appreciation of the limitations imposed by the method, the work will be of great value. G. B.